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Course in Postgraduate Gastroenterology
The Shoreham and Walter Reed Army Hospital
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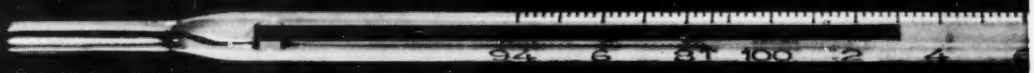

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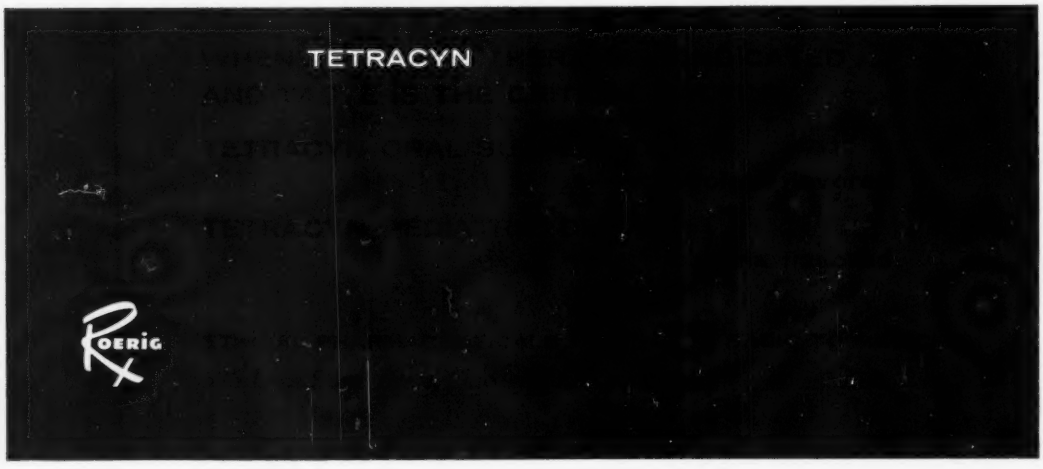

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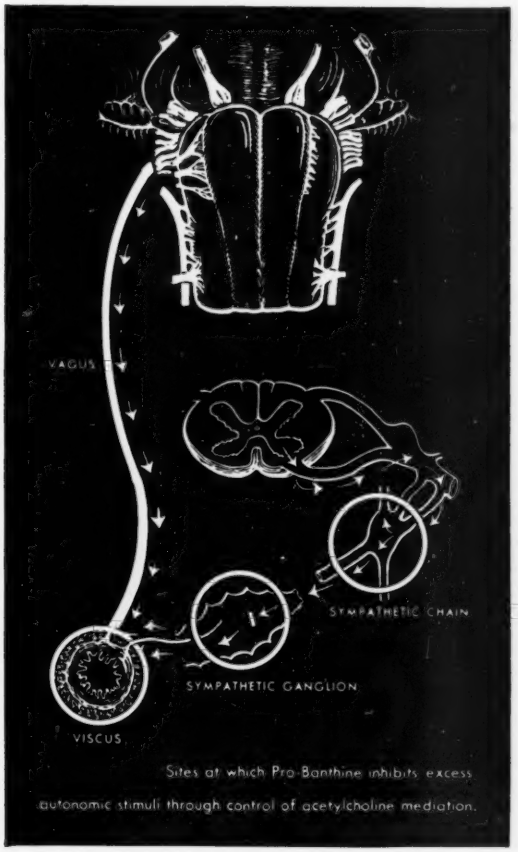

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1. Schwartz I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: Gastroenterology *25*:416 (Nov.) 1953.

2. Roback, R. A., and Beal, J. M.: Gastroenterology *25*:24 (Sept.) 1953.

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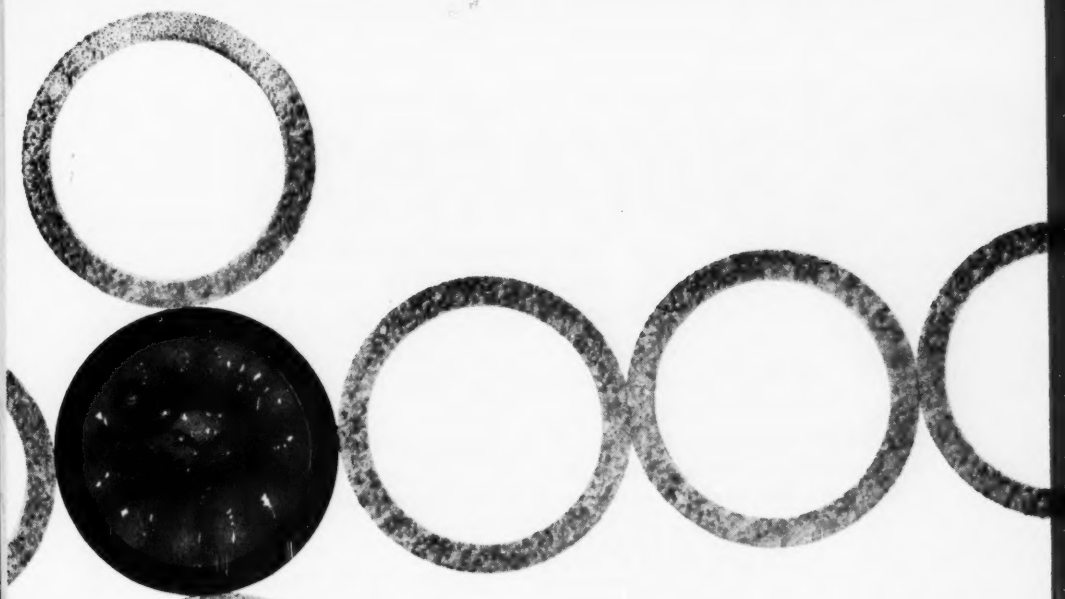

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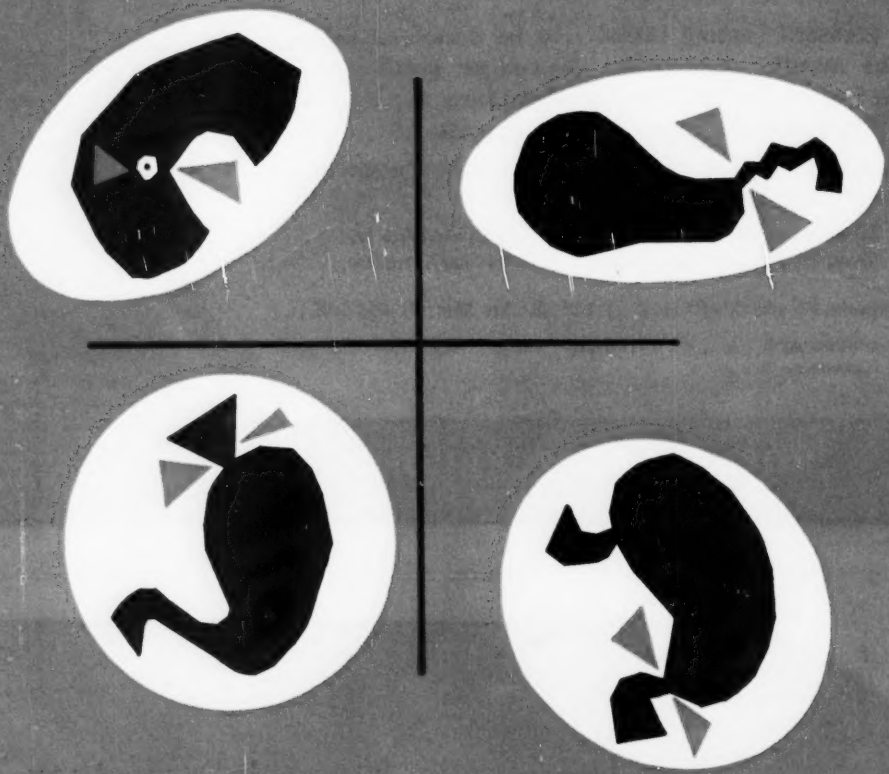

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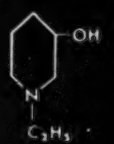

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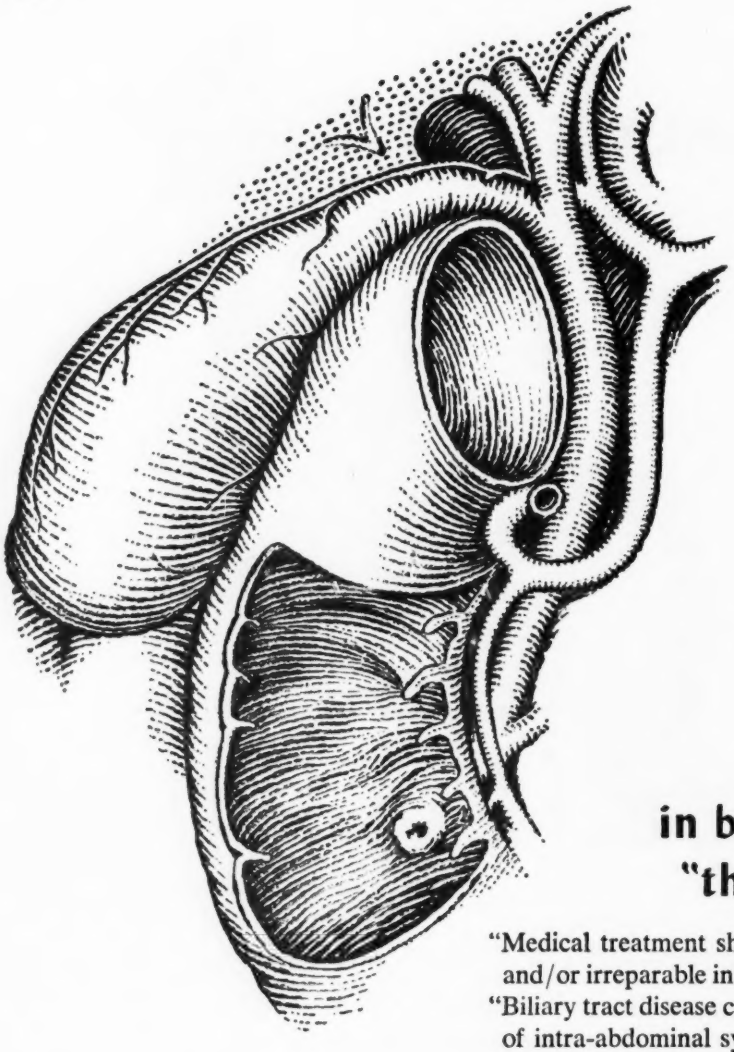

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VOLUME 22 SEPTEMBER, 1954 NUMBER 3

CONTROL OF STOMACH PAIN*

DONALD C. BALFOUR, Jr., M.D.†
Los Angeles, Calif.

The ways and means of controlling pain arising in the upper gastrointestinal tract is a subject often assigned for review because the symptoms are common and management is difficult. Although the purpose of this paper is to discuss the therapeutic measures available in medical treatment, it is basic that the causes responsible for the symptoms and the mechanisms by which the pain is produced be evaluated. In view of this fact a brief summary will be made of our present knowledge of these fundamental factors.

TYPES OF PAIN

Three types of pain may arise from the upper gastrointestinal tract. These are: (1) Visceral pain, e.g. tightness, anorexia, nausea, burning, cramping, or dyspepsia, (2) referred pain; e.g. guarding, spasm, and pain on the body wall and (3) somatic pain; e.g. gnawing, constant, boring pain which radiates to the back, chest, or abdominal wall.

Visceral pain may be caused by (1) psychogenic reactions, (2) mechanical or chemical trauma, (3) heat or cold, (4) inflammation, (5) distention or (6) new growths. All abnormal sensations or manifestations of pain that arise in viscera are caused by smooth muscle irritation in the visceral wall which in turn send stimuli over afferent nerve fibers. Any spasm, stretching by pressure, or increase of tone of a portion of the musculature will be a source of some type of a stimulus. When the smooth muscle fibers of the visceral wall are chronically inflamed or irritated they will be sensitive in a way similar to other muscles in the body. Thus the symptom of morning nausea may be a manifestation of muscle irritation just as morning stiffness and soreness can occur in other muscles. The true character of visceral pain as a reaction to heat and cold

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†Assistant Clinical Professor of Medicine, University of Southern California, School of Medicine, Department of Medicine (Gastroenterology).

is believed to be a soothing effect of warmth on the muscle in the case of heat, and spasm of the musculature in the case of cold. Any extremes of heat may, however, irritate the mucosa to such an extent that the musculature will be stimulated by reflex spasm more than the warmth may soothe. Other lesions of the mucosa, including erosion of ulcers to free nerve endings, do not cause visceral pain except by causing reflex spasm in the muscle wall.

Referred pain is a sensation of pain felt superficially on the body wall. This is a sensation of real pain which can be present even when there is no visceral pain but only digestive symptoms. The referred pain arises from the visceral symptoms by the effect of stimuli over sympathetic nervous system fibers irritating adjacent cerebrospinal neurons in the spinal cord. The areas of referred pain of the esophagus, stomach and duodenum are in the epigastrium but are not well enough localized to determine the exact source of the pain in the viscera. Referred pain may be relieved by local anesthetization of skin and muscle and the patient may be relieved of practically all pain by this procedure[1]. What visceral sensations are present, however, will persist.

Somatic pain is caused by lesions of the upper gastrointestinal tract when the inflammatory reaction, edema, pressure or penetration of the lesion has extended to the parietal peritoneum or into the mesenteries. The deep boring constant somatic pain will radiate to the back, chest or abdominal wall. Where this pain is felt is important in localization of the lesion, and it means that the lesion has extended through the visceral wall.

Pathways of Pain

Three pathways of pain from the upper gastrointestinal tract are possible: (1) The vagus nerve, (2) the sympathetic nervous system and (3) the cerebrospinal nerves of the abdominal wall. All of these nerve pathways have afferent fibers but only the sympathetic system and the spinal nerves distributed to the abdominal wall carry afferent pain fibers below the diaphragm. The vagus nerve carries no pain fibers in the abdomen and the relief of pain by cutting or blocking the vagus nerve is a result of a change in motility[2]. The afferent visceral pathways travel over the sympathetic nervous system via the splanchnic nerves. The somatic pain is carried over somatic nerve fibers which go to the parietal peritoneum and extend part way into the mesenteries. The phrenic nerve carried pain from irritation of the dome of the diaphragm and the pain is felt in the shoulder. This unusual pattern of pain is due to the unusual distribution of the phrenic nerve. It is not a true visceral-somatic referred pain.

Pain as a Stress Reaction

With the increasing stress in our every day living and more indiscretions of eating, drinking, and smoking, it is to be expected that symptoms in the upper gastrointestinal tract would be more common. It is now known that there are two different pathways by which stress on an individual can affect

the stomach. A combination of these factors can bring about irritative changes in the stomach or the esophagus or the duodenum.

To mention one of these problems let us first consider emotional stress. The reaction pattern of the individual is all important. If the individual has a reaction pattern of fear, or resentment, and wants help in facing the emotional problem or tension, the distress will probably react on the upper gastrointestinal tract in contradistinction to the lower gastrointestinal tract. There are two pathways by which this distress reaction can affect the stomach. It has been recently shown that reaction stimuli going primarily to the anterior hypothalamus will affect the stomach primarily over the vagus nerve, whereas reaction patterns affecting the posterior hypothalamus will affect the stomach by way of the pituitary-adrenal hormonal pathway[3]. The vagal pathway causes sudden changes in motility and acid production. The hormonal influence of the stimulated adrenal will cause a prolonged persistent increase in the secretions of the stomach. Reactions seen in the stomach following physical trauma to the body such as a broken bone or severe burn are believed to be caused by the adaptation syndrome or the stimulated adrenal glands.

Some of the evidence that we have to justify these conclusions is as follows: With our knowledge of uropepsin we have been able to see how some of these reactions work on the stomach[4]. Stimulation of the vagus nerve causes high excretion of acid but does not cause elevation of uropepsin excretion. A sudden great increase in the motility of the stomach by stimulation of the vagus nerve may cause a rise in uropepsin excretion for a short time. A constant high output of uropepsin, however, is brought about only by overactivity of the adrenal glands. The hormonal effect is more pronounced in the male than it is in the female because the androgenic hormones which are higher in the male have a tendency to stimulate secretion in the stomach. In the future when these tests are more generally used, a better understanding of the responses and the mechanisms by which each individual overstimulates the stomach should be possible. A lower uropepsin level with high acid response should indicate that the problem was primarily stimulation over the vagus nerve whereas a high uropepsin level would indicate hormonal influences on gastric secretion. Again it should be emphasized that it is a combination of all factors that determines whether or not these stress reactions will cause irritative lesions to develop in the stomach, duodenum or esophagus.

This discussion will not include the problems of differential diagnosis related to cancer or the intractable pain of benign lesions that can only be treated by the surgeon. It can be hoped, however, that as patients now come for advice sooner than they did in the past, many intractable benign lesions can be avoided.

Control of Tension

Little can be said in regard to control of tension in an individual other than the most important factor of the physician taking a sympathetic and under-

standing attitude in regard to the causes of tension. Usually the tension is a matter of personality reaction or habit which cannot be tolerated with the advancing of age. Statistics shown in regard to peptic ulcer suggest that when habitual tension is stress enough to cause an ulcer before the age of twenty, the lesion will probably become intractable[5]. The patient should not be allowed to have any fear or resentment of their problems nor should any state of indecision be permitted any longer than is necessary. A great deal of reassurance is necessary for this. An explanation of the truth about tension as being a habit developed as a normal reaction to circumstances is usually very helpful. When a patient realizes this fact, he will have less anxiety about himself.

It would be wise to mention the use of sedatives in controlling tension. The average patient will find that they can control their tension when they understand it. When this cannot be done, however, sedatives have to be used. Best results can be obtained when sedatives are used sparingly and only when necessary. The patients learn to know when they are tense and can regulate the use of sedation themselves. It should be mentioned here that sedation strong enough to have a profound effect on tension is usually irritating to the stomach. Anytime that the stronger sedatives are used during the day or at bed time for sleep it should be emphasized that they be taken with a soothing food or counter-irritant.

Control of Pain by Diet

One hundred years ago Abercrombie, writing about the treatment of upper gastrointestinal distress, emphasized the fact that the volume of food was perhaps the most important feature of dietary management. No one has contradicted this opinion, and the fact that so many diets have been advocated emphasizes the fact that the type of food eaten is not as important as is the amount eaten at one time. Small frequent feedings are most important.

General considerations about the type of foods that should be eaten to control pain include several points of interest. We are more aware now of the foods that stimulate gastric function and with the more common use of these foods and drinks it is important to insist that they not be taken. Adequate protein intake is necessary but we have evidence that excessive protein intake brings about increased gastric function[4]. Therefore, the concentration of protein in the diet should be adequate but not excessive. Fat intake should be as high as is consistent with the desired caloric intake but the fats should be of a low melting point such as dairy or vegetable fats. They are soothing to the gastrointestinal tract and decrease gastric motility and secretion by causing the production of enterogastrone. A few ounces of olive oil will be especially soothing to the acute ulcer. Liquids should be taken between meals and not at meal time. Washing the food down with water or liquids has a detrimental effect upon digestion and usually causes more swallowing of air. Regardless of what feeding or medicinal program is used, 10 glasses or more of tepid water should be taken during a day.

As it is important to bathe lesions on the surface of the body, it is also important to bathe the irritations that are on the mucosa of the esophagus, stomach, or duodenum.

All foods are antacids except a few of the acid fruits which should be used with caution. Neutralization of the excessive acid production seen in irritations of the stomach, duodenum, and esophagus is important. Therefore, food may be used as an antacid as much as the caloric intake will permit. Commercial antacid preparations may be used when the caloric intake must be limited. Sodium bicarbonate is one antacid which has a specific antispastic effect that may explain its common use. It is not as strong an antacid as many other chemical compounds[6].

The foods that are used when there is irritation of the upper gastrointestinal tract should be easy to digest. There should be little roughage or solid food that is difficult for the stomach to make into a soft chyme. Milk is not easily digested in the intestines and its residue is slightly irritating to the bowel. Changing the proteins by boiling the milk before eating is helpful when this problem causes symptoms. We have observed that the use of a soy bean milk (Mull-soy, powdered)* instead of cow's milk has been noticeably more soothing to the upper gastrointestinal tract and seemingly easier to digest. The taste of the powdered soy bean milk is tolerable but precludes its use in some cases.

No matter what other treatment is used in the control of peptic ulceration or irritation of the upper gastrointestinal tract, the wise and careful use of diet must be accomplished. Other measures of treatment may control pain temporarily but the patient may develop more trouble because the lesion progresses unless the diet is proper for healing.

Blocking Nerve Fibers

The atropine-like drugs have been used for many years to prevent neurogenic stimulation of the stomach by blocking the vagus nerve. Many improved drugs have been developed and many other blocking agents will be presented to the medical profession in the future. A few of the specific accomplishments that should be expected of these medicines can be mentioned. The medicine should not be irritating to the mucosa of the stomach and usually it is possible to evaluate this by holding a tablet in the mouth. Both a vagal and sympathetic blocking effect is desirable. The medicine must accomplish dryness of the secretions of the mouth in order to accomplish dryness in the secretions of the stomach. The medicine that will do this and not cause increased sweating, weakness or palpation of the heart would be ideal. Such a medicine has not as yet specifically accomplished all these effects but there are many that will accomplish profound blocking effects without side reactions. It must be remem-

*Borden Company, New York, N. Y.

bered, however, that in those cases with a high uropepsin suggesting marked hormonal influence, blocking the nerves may not adequately decrease gastric secretion.

Anesthetizing Nerve Ending

For many years it has been known that topical anesthetics can be used to anesthetize pain in the upper gastrointestinal tract. It has only been in recent years, however, that this method of treatment has been developed to where it can be used effectively and without danger[7]. One reason for this delay in the use of topical anesthetics is because the first anesthetics used were habit forming drugs and their use only in advanced malignancy seemed justifiable. With the development of many new nonhabit forming and nontoxic topical anesthetics, more general use of this method of controlling pain is possible.

Several general principles will be mentioned here. The anesthetic solution must come into contact with the lesion and be in contact with the lesion long enough to anesthetize the surface. For esophageal irritation the anesthetic must be held in a cohesive vehicle which will remain on the walls of the esophagus long enough to create anesthetization. Watery solutions may be used for stomach irritations or duodenal irritations and, if there is no food or mucus preventing contact with the lesion, relief of the most severe pain can be accomplished. It is important to mention here that mild visceral irritations causing nausea or tightness can be relieved by this anesthetization effect. The use of this medicine has suggested many of the mechanisms of pain mentioned earlier in this paper.

Many substances have an anesthetic effect. Some of the antihistaminic preparations and a few of the preparations used in ulcer therapy have relieved nausea or distress or dyspepsia because of their anesthetic quality. A nontoxic topical anesthetic such as procaine hydrochloride can be used to even better effect. Procaine hydrochloride can be mixed with a cohesive substance such as methyl cellulose for esophageal lesions or the plain liquid can be given for lesions of the stomach or duodenum. A standardized liquid preparation of 10 per cent procaine hydrochloride (Naucaine)* is commercially available with flavoring. One teaspoonful to one tablespoon of this preparation is convenient for use. Xylocaine† is being put out commercially in a methyl cellulose base. This being a stronger anesthetic, a 2 per cent solution gives excellent relief of esophageal irritation and the mixture is quite effective in the stomach and duodenum.

Tablets are being used (Naucaine) and medications containing an anesthetic in the capsule (Procules OH)‡ and capsules. These solid preparations are not as effective as the liquids although they will be satisfactory in many

*Taylor Laboratories, Inc., Houtson, Texas.
†Astra Pharmaceutical Products, Inc., Worcester, Mass.
‡Cutter Laboratories, Berkeley, Calif.

cases of milder irritations in the lower stomach and duodenum. The efficacy with which the solid material is dispersed into solution determines the effectiveness of the medication. It must be remembered, however, that in using this medication to control pain just as with the nerve blocking agent, complete control of the diet and tension should be accomplished to achieve lasting results.

Control of Pain After Stomach Surgery

After gastric surgery when the food falls immediately into the small intestine several variations in the above discussion for control of pain must be followed. Briefly these changes include the variation of the diet to a relatively dry diet that will not rush through the small intestine. Extremely careful regulation of temperature along with a moderate control of the osmotic pressure of any of the food or liquid is essential[8]. Frequently, it is found that a continuation of the previously successful ulcer diet will cause many distressing symptoms after gastric resection. When the cause of symptoms is due to jejunitis in the afferent loop, the topical anesthetics are especially useful[9].

Summary

The control of pain arising in the upper gastrointestinal tract is reviewed with special mention of methods of measuring stress effect and the use of anesthetic agents.

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Discussion

Dr. I. Snapper:—I have listened with great attention to Dr. Balfour because dietetic therapy depends so much upon the country a person lives in and so many different diets work differently in different countries.

Around 1900 the pure milk diet of Leube was the most popular method for the treatment of ulcers. Before 1910 Lenhartz advised against the milk diet but advocated a much richer diet, identical to the modern Meulengracht diet. He was succeeded by Sippy whom all of you know. Now most physicians prescribe a Meulengracht diet in which milk and cereals are given only for a very short time before a more extensive diet is given. This is a point of greatest interest. The Leube milk diet was succeeded by Lenhartz' diet because a certain number of patients continued bleeding with the Leube diet. Then Meulengracht for the same reason preferred his diet over the Sippy diet. This, however, was also the reason why the Lenhartz' diet was abandoned for the Sippy diet. It follows that certain bleeding patients will do better on a Meulengracht diet than on a milk diet, others better on a milk diet than on a Meulengracht diet. Often a consultant is invited because an ulcer patient continues bleeding. Blood is being poured in from both arms, the surgeons are already whetting their knives. There is an excellent way of preventing the operation. If the patient had been given a Meulengracht diet then change this diet for milk diluted with lime water. If the patient has been on a milk diet change for a Meulengracht diet. When at the same time the amounts of blood transfused are cut down to the minimum sufficient to ward off signs of shock, then in my experience it is nearly always possible to avoid the operation during the hemorrhage.

I merely bring this out in order to show how much difference there has been in dietary concepts in the course of the years. In 1920 we smiled about our ideas of 1900, but in 1950 we have a completely different concept than we had in 1920. In 1980 we probably will smile again about our 1950 ideas. Thus, we must try to remember that even today the last word about our choice of dietary or surgical treatment has not yet been spoken.

Diets are very much dependent upon local habits. In this country, where ice cream is an important part of the diet, ulcer patients are allowed, sometimes even encouraged, to eat ice cream. In other countries it was always held that neither excessively hot nor excessively cold nutrients should be eaten. Nevertheless many of our patients are at least not harmed by eating ice cream.

Lamb chops are a popular item for ulcer diets in this country but, in the rest of the world lamb chops are considered to be heavily digestible. Veal is given in Europe for ulcers but it is considered to be undigestible in this country.

Dr. Balfour correctly emphasized the necessity for searching for a certain amount of rationale in prescribing a diet. Advisable as this may be I am afraid that for the time being at least, the Hippocratic empirical method is the only way to find out which ulcer diet fits a special patient best.

Dr. Balfour is very much interested in psychogenic influence upon ulcers. In my opinion, psychosomatic influences play a very important role even in dietetic therapy. I can understand that with correct psychological sedation, like

Dr. Balfour described, even lamb chops and ice cream, may be the right diet for an ulcer patient.

Dr. Balfour mentioned the research of Dr. Emanuel Libman about referred pain. Libman contended that the regions of referred pain were different depending on whether the patient was hypersensitive or hyposensitive. If the patient could stand pressure upon his mastoid process without too much discomfort he was hyposensitive, if a patient reacted already upon slight pressure he was hypersensitive. In Libman's opinion hyposensitive patients with an ulcer of the duodenum often felt the pain in the lower parts of the abdomen, usually at the left side.

Dr. Balfour:—In my experience I have not been able to make any deductions here and I wonder if anybody can make any deductions about the hyposensitive patient. Frequently such a patient will not be too aware or be too upset by the referred pain stimulation as it is normally felt in the epigastrium. When the pain moves downward in the duodenal area there is a deep tenderness and soreness. Then the pain will be felt in the lower epigastrium or possibly even in the back. I think then we are getting into the somatic pain. In the hyposensitive patient with the more severe somatic pain from the edema and the inflammation, then the referred pain will be almost overlooked and not appreciated.

Dr. O. H. Wangensteen:—I was very much interested in Dr. Balfour's presentation as you were. Dr. Balfour's father spent his life doing gastric resection for what Dr. Balfour, Jr. now finds can be treated successfully by diet and by advising with the patient. If ulcer relief can be accomplished that readily, I think I, too, would go along with the idea. The most effective treatment achieved by the simplest means is the best way.

Oftentimes, spoken words are as sweet as honey in the mouth but bitter in the belly. I think that is perhaps true of some foods ingested by patients with the acid-peptic ulcer diathesis. Dr. Balfour said a great deal about what foods to take, but he said nothing about what foods not to take. Even surgeons (prevailing opinions to the contrary), have to be very attentive to what their patients say. I have learned to be a good listener in administering to patients even though I am not a psychiatrist. I would say that coffee is number one on the list of things that patients who have a peptic ulcer should not take. Long years ago Dr. K. A. Merendino, now at the University of Washington in Seattle, and I worked together on the problem of ulcer provocation with caffeine. Dogs, unlike man are quite unresponsive to caffeine. When Dr. Merendino gave dogs 2,500 mg. of caffeine, he was able to demonstrate evidence of stimulation of acid secretion following the administration of caffeine. I have the impression that if a patient is to be treated medically for peptic ulcer, he should not take caffeine, in any form whatsoever. Man must be a thousand times more sensitive to caffeine than is the dog.

St. Paul said, "Take a little wine for your stomach's sake". I don't know whether Drs. Balfour and Snapper would agree with St. Paul's proposition for a patient with peptic ulcer. In listening to patients, however, I find that of alcoholic drinks, beer appears to be the worst of all. Just why, I don't know, but many patients with an ulcer have told me they cannot take beer.

I believe the general opinion is that smoking is pretty bad for a patient with an ulcer. We saw Dr. Bachrach's demonstration of the effect of nicotine upon the motility of the stomach. Dr. Robert Toon in our Experimental Laboratories did a very interesting experiment with cigarette smoking in the dog. He used a long cigarette holder in dogs which had tracheostomies. The dogs would puff away on the cigarettes without burning themselves. By having dogs smoke about five cigarettes a day which is perhaps a lot of smoking for a dog, Toon was able to show that nicotine potentiated the ulcer diathesis. Denicotinized cigarettes were without effect.

I would say that these are three "don'ts" for patients with peptic ulcer. Dr. Balfour went over quite satisfactorily the things that ulcer patients should do. He did not say anything about the long interval of night starvation. I have often said that if a patient with an ulcer awakened himself with an alarm clock and took some milk during the night, and ate before going to bed, and again in the morning, directly on awakening, the long empty period for the stomach at night would be avoided—an important item in the acid-peptic ulcer diathesis. As Dr. Balfour was talking about putting the patient with a peptic ulcer in the proper frame of mind, I could not refrain from thinking that Dr. Balfour in the role of Diogenes might have an easier time to find an honest than a happy man.

I suppose a surgeon should not expose his ignorance concerning attempting to relieve all these tensions of life by trying to analyze the problem, for it seems to me it is almost insoluble. What can you give a man to make him happy? You say you can give him everything he wants, but would he be happy? Man's wants are about as numerous as the stars in the sky or the sands on the beach. Whether a patient with a peptic ulcer, who could have his immediate wants and needs sugar coated with all of these nice things, which Dr. Balfour would do for him—whether the patient's internal tensions would yield and remain subdued long beyond Dr. Balfour's ministration is an interesting question. I believe that an internist with a broad gauged view who will look at some of these problems realistically like Dr. Balfour does—such a physician is probably more likely to be able to resolve the patients' anxieties in a more acceptable manner than the out and out analyst or psychiatrist, who is more likely to give the inquiries ultra oblique directions.

There is one thing about an ulcer diet upon which I have laid great emphasis. I want to deplore the prevalent tendency on the part of many internists to give a mixture of half milk and half cream to peptic ulcer patients. Under this

regimen, many a patient gets fat and still retains his ulcer. It is the protein in the milk which has the important combining value for neutralizing acid. Skim milk contains as much protein as cream. A patient can take considerably more skim milk than whole milk or cream. And as the pH of the gastric juice is raised by skim milk, the peptic activity of the gastric juice also is decreased.

In patients with a bleeding peptic ulcer, I like to insert a small polyethylene catheter into the stomach through the nose and drip skim milk 24 hours a day. If the bleeding does not stop under this therapy, the patient should be operated upon. Of all the substrates which my associates and I have tried to raise the intragastric pH, skim milk is definitely the best.

The weight of peptic ulcer patients, who come to operation, is a question of no small interest to the surgeon. Internists, too, should be more alert to this important matter. What good is served by having a patient with a peptic ulcer get fat? I have seen no evidence that obesity helps to overcome the ulcer diathesis. Skim milk is a far better regimen for such patients to control acidity and pain than is a mixture of half milk and half cream. In fact, I frequently employ a skim milk regimen of 3 liters a day, supplemented only by Vitamin C and iron to correct the obesity of fat patients with peptic ulcer before accepting them for operation. The patient who accepts the diet does lose weight. Moreover, many a patient is astonished to find how well his ulcer does on this regimen. There are few foods which are superior to skim milk in the control of gastric acidity. In fact, I know of none.

Dr. Balfour:—Thank you very much, Dr. Wangensteen. I am sure we are all familiar with these "do" and "don'ts". Coffee combines the worse evils and it certainly is also irritating to the mucosa. The general principles I mentioned are an attempt to try to get to the common factors.

The other comment is also basic. Too much concentrated proteins, I will again mention, will cause an increase in gastric function. Plain skim milk is just barely an adequate protein diet during the day so it will not increase gastric function and can be used as an excellent treatment in the usual problems for this situation.

ACUTE ABDOMINAL DISORDERS*

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The subject of acute abdominal conditions is of special interest to both gastroenterologists and general surgeons. It is also the concern of pediatricians, obstetricians, psychiatrists, gynecologists, cardiologists, allergists and others. The names of Alvarez, Zachery Cope, Brennemann, Wangensteen, Bockus and many others are associated with basic contributions to this subject in recent years. The general use of antibiotics, blood transfusions, the advances in the understanding of electrolytes, the use of new laboratory technics and the introduction of cortisone and ACTH in clinical medicine have altered older concepts of the acute abdomen and justify a new presentation of this old subject. For instance, it is important to realize that a patient who has been receiving cortisone over a long period of time, and then needs an emergency surgical procedure must be given additional large preoperative dosages of cortisone to avoid irreversible shock following surgery[1].

The most practical approach to a discussion of this subject is to pick out the seven most fundamental conditions which, as clinicians, we must attend. A study of the discharge diagnoses at the Hollywood Presbyterian Hospital during the five year period ending 1952 (Table I) revealed acute abdominal conditions in their order of frequency as follows: Appendicitis, female pelvic disease, intestinal obstruction, gastrointestinal bleeding, acute pancreatitis, and perforated peptic ulcer.

Acute Appendicitis:—The early diagnosis of acute appendicitis is now made with much greater frequency than a decade ago; however, it seems true that in recent years the general use of antibiotics has tended to minimize the usual signs and thus postpone recognition[2]. This is especially true in children, who are often given an antibiotic for suspected influenza, enteritis, or acute mesenteric adenitis. This can, and has delayed the diagnosis of obstructive appendicitis with resulting rupture.

Appendicitis is rare in infancy; however, 447 proved cases were recently reported[3] from the world's literature. From the second year on, it occurs with increasing frequency throughout childhood. As has been previously mentioned, respiratory infections and enteritis are especially deceptive in this age group. The unrecognized fact that diarrhea may well accompany acute appendicitis in children has led to a great number of professional mistakes and disastrous delays. The telephone report that the child has a diarrhea is not an indication for more antibiotics, but for examination.

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In the aged, the minimal reaction often caused by appendicitis must be kept uppermost in the examiner's mind. A recent 83-year old woman had only slight indigestion and questionable abdominal findings for three days before a gangrenous appendix was removed.

Right lower quadrant findings can be confused with stone in the ureter, pyelitis, hydronephrosis, and seminal vesiculitis[4]. The high incidence of McBurney incisions in patients with urinary stone is mute evidence alerting us to this pitfall. Intravenous pyelograms have been stressed as a necessary part of the emergency differential in questionable cases[5]. Ovarian dysfunction with ovarian pain produces a picture difficult to differentiate from early mild acute appendicitis. In a recent study by Patton[6], 79 per cent of the appendectomies for chronic appendicitis had recurrent trouble. In a study of 50 patients with hysteria, by

TABLE I

ACUTE ABDOMINAL DISORDERS
HOLLYWOOD PRESBYTERIAN HOSPITAL 1947-1952: TOTAL 637

Entity	Number	Percentage
Acute appendicitis	289	45
Female pelvic disease	154	24
Intestinal obstructions	85	13
Gastrointestinal bleeding	40	6
Acute cholecystitis	35	5
Acute pancreatitis	19	3
Perforated ulcers	15	2

Cases of acute appendicitis were all proved pathologically. Female pelvic disease included salpingitis and ectopic pregnancy. Gastrointestinal bleeding included those of moderate severity. Diagnoses were obtained from discharge notes and did not include cases suspected at entry.

Cohen[7], it was found that women between the ages of 30 and 34 had 60 per cent more operations than men. In 50 hysterics, 68 per cent had had an appendectomy performed. Aids in suspecting the diagnosis of hysteria and avoiding surgery are a long history of multiple complaints, the usual bizarre and atypical finding and the lack of constitutional reaction in temperature and white blood count. Here an intimate knowledge of the patient and the willingness to observe at four hour intervals with an open mind may be of much greater service to the patient than exploration. It should be recognized by all that when the surgeon is called in, it may still be wise to withhold an impression and delay action. Internists and surgeons together must be willing to assume such a responsibility even though this course is not easy to explain to the patient or his relatives.

Other conditions causing right lower quadrant findings are salpingitis, twisted or ruptured cyst, ectopic pregnancy and slow drainage from a perforated ulcer into the right gutter.

Left lower quadrant findings, in middle-aged or older patients, suggest diverticulitis. Here an x-ray for location of the cecum may be important for differentiating left-sided appendicitis. This may be an important differential as most cases of acute diverticulitis can be treated properly with conservative measures. With perforation; however, and early generalized spread of the peritonitis, exploration, drainage, and at times, transverse colostomy will be indicated.

With high abdominal findings, differential between gallbladder disease and appendicitis may be difficult. Pancreatitis, penetrating ulcer, hepatitis, and kidney disease produce findings in this area.

There are many general conditions which produce signs suggestive of appendicitis. Upper respiratory conditions, influenza and enteritis have been mentioned with regard to children. At the onset, poliomyelitis, rheumatic fever, and the exanthemata may give abdominal signs. A child with the story of indigestion, with right lower quadrant findings and without any other evidence of poliomyelitis including a negative spinal tap had an appendectomy and two days later came down with full fledged poliomyelitis. Such an experience makes one cautious and eager to impart this caution to others. Early tetanus may, especially, involve the abdominal muscles. Black widow spider bite, leutic crises and sickle cell anemia, at times, present a problem. If one will check all patients with abdominal pain for a lead line and think of red cell stippling, he will occasionally be rewarded. Studies by Dunphy[8] and McClenahan[9] have shown that many cases with mesenteric thrombosis have a long history of intermittent abdominal pain. From these studies has come the concept of abdominal angina in which lesser episodes of arterial spasm are a reasonable explanation for many transient abdominal pains. Abdominal migraine or epilepsy is usually followed by lethargy and electroencephalographic changes.

As regards treatment, once the diagnosis of appendicitis is made operation is usually indicated. Antibiotics, blood, and electrolyte replacement have extended the indications for early surgery even in the most serious cases. The Ochsner method of management usually is now considered advisable only for a number of hours in preparation for surgery. Well walled-off appendiceal abscess may be an exception to this dictum. With antibiotics, Wangensteen suction, supportive fluids, blood, and by utilizing aspiration at the time of surgery to remove any spillage of pus, makes it possible to remove safely the offending organ in almost all cases. This avoids prolonged sepsis and secondary operation.

Acute Pelvic Disease:—Ovarian pain has already been referred to in relation to appendicitis. In the acute abdominal conditions, the pelvic organs play the second most frequent role. Hysteria in women, with an onset between the ages of 10 and 35 is recognized by the astute diagnostician. Acute salpingitis appears

to be considerably less frequent on the wards of the County Hospital in recent years, which is probably the result of the wide usage of antibiotics. The differentiation between this and pelvic appendicitis; however, at times is impossible and since therapy with antibiotics is hazardous for the former, appendectomy is necessary. Perforated or twisted ovarian cysts require early surgery. Recognition is usually possible with bimanual pelvic examination. Pelvic pain, a skipped period, spotting, tenderness and pelvic mass suggest ectopic pregnancy. Spontaneous hemorrhage into the rectus msucle may be confusing as it, too, can form a pelvic mass by pushing the pelvic peritoneum before it, into the pelvis[10].

Intestinal Obstruction:—The colicky pain of intestinal obstruction directs our thoughts to this condition. So does a scar on the abdomen. In the newborn, vomiting, soft distention and a flat film are the triad for diagnosis. A little later, persistent colic and a bloody diarrhea suggest intussusception. In the adult, typical colic may not be prominent in large bowel obstruction and enemas for a time may bring some results. Here, again, the flat film is essential. In both small and large bowel obstruction, the repetition of the flat film at frequent intervals[11], while under observation, is important as the x-ray evidence may be slow to develop. The most difficult type of intestinal obstruction to recognize may be the twisted loop, yet this is just the variety in which early recognition is most important. Abdominal tenderness or pelvic bimanual tenderness is usual if repeatedly sought for. Elevation of the white blood count in this type of obstruction is frequent. Two special types of obstruction are intriguing; gallstone ileus may give little abdominal distention for several days. If the onset simulates acute cholecystitis and a suction tube is put in place to relieve vomiting, the diagnosis may be missed until a later stage. Aids in avoiding this error are the simple listening to the abdomen for the almost pathognomonic high pitched peristaltic tinkle and also the x-ray films which clearly show the occasionally unsuspected obstruction. Strangulated femoral hernia is often missed because it is small and because a concerted effort to feel for it is not made. When the condition has gone on for four or five days, in an elderly person, a critical situation arises and the mortality under these circumstances has been extremely high. A few years ago, Gatch[12] suggested a novel method of surgical management which has been extremely helpful. In extremely ill patients, seen late, he suggested simply incising the gangrenous femoral loop, decompressing the dilated small intestine with a Miller-Abbott tube and doing an interval intestinal operation several days later. The value of this surgical trick should be recognized more widely.

Treatment of the twisted loop usually requires surgery within a few hours though preparation with suction, blood and electrolytes is necessary for the depleted patient. Antibiotics have been proved both experimentally and clinically to be of great additional value in the management of these cases. In simple obstruction, adequate decompression with Wangensteen suction and the two lumen tube accompanied by satisfactory replacement of sodium, potassium, and

chlorides and water until good urinary output is demonstrated over a period of hours has made surgical relief much safer than in years gone by. The basic contribution by Dr. Wangensteen, of utilizing intestinal suction for decompression in the relief and preparation of patients with intestinal obstruction is one of the oustanding contributions in this field, and will not be forgotten.

Gastrointestinal Bleeding:—Gastrointestinal hemorrhage has not conventionally been considered under acute abdominal conditions and yet, with the present day emphasis on early abdominal surgery for selected cases of massive bleeding from peptic ulcer, it seems proper to include it. At the present time there is pretty general agreement that patients with massive bleeding from peptic ulcers, who do not show a continued good response to about eight pints of blood, given during the first twelve hours, or who show further signs of bleeding after its administration, are best considered as failing to respond to medical management and should be prepared for surgery[13]. Availability of adequate amounts of blood, proper operating facilities and a competent experienced surgeon are essential before resorting to this form of treatment. All the facets of the individual case must be evaluated and this situation is surely best handled, in the interest of the patient, by close cooperation of the gastroenterologist and the surgeon.

Acute Cholecystitis:—The diagnosis of acute cholecystitis and gallbladder colic is usually not so difficult as the decision for its proper management. The aggressive surgical attitude utilized by Glenn[14] for almost all cases is opposed by Ogilvie[15] and others[16]. Perhaps, a middle of the road policy and individualization of cases would be safest. When the signs and symptoms of perforation are suggestive, or in good risk cases with early disease, with a well equipped surgery and surgeon, cholecystitis can best be treated by early surgery; otherwise, conservative therapy and interval surgery may be wisest. Furthermore, there is no substitute for good judgment in the operating room where cholecystectomy or some lesser procedure must be decided upon. The hazard of injuring the common duct, or hepatic artery in acute disease makes many surgeons prefer to delay operation whenever this course seems safe.

Acute Pancreatitis:—The diagnosis of acute pancreatitis has become, during the last ten years, as simple and accurate as that of stones in the gallbladder. Almost every admission to the hospital with a pain in the upper abdomen, not obviously explained, has a blood amylase test and this is as it should be. The advantages of the nonoperative treatment of this condition in most cases has been pretty well demonstrated. A high amylase level, per se, however, does not mean that conservative measures will always suffice. A recent patient with upper abdominal symptoms and a 1,600 blood amylase was, after much delay, operated upon and an unsuspected gangrenous loop of intestine found at operation. In another recent case, a patient a few days after an appendectomy developed upper abdominal pain and vomiting. Blood amylase determinations were as high as 1,200. Suction and atropine were administered and, after a few days, the tube

was removed with recurrence of symptoms. X-rays of the abdomen were not revealing. Further amylase levels were high. Finally, operation was performed and a nearly complete simple obstruction of the ileum was relieved. The amylase levels dropped to normal following surgery. Further studies to verify the frequency of this occurrence must be made, but it is enough to alert one to the hazards of complacency based on a high amylase level.

It is well known that penetrating duodenal ulcer, perforated ulcer and salivary gland disease may produce high amylase levels, so it behooves us as clinicians to carefully evaluate each case as in the days before the blood amylase determination was available. To consider a high amylase level in a patient with upper abdominal pain as synonymous with pancreatitis in all cases will court an occasional disaster.

Furthermore, the reported mortality for pancreatitis treated conservatively is statistically too high (21 per cent in a recent large series[17]) for complacency. Joel Baker, of Seattle, recently has raised the question of surgery in some cases of pancreatitis with elevated bilirubin on the possibility that removal of a stone might be considered as an emergency procedure. Dr. Jonathan Rhoads in 1949[18] went even further, doing a choledocostomy and drainage down to the pancreatic capsule in most cases. His results were remarkably good; that is, 3 per cent mortality in 31 cases. He attributed part of his good results to the use of penicillin. The use of aureomycin in experimentally produced pancreatitis in dogs has reduced mortality 90 per cent[19]. Perhaps more antibiotics and even occasionally surgical intervention will again have a place with the more severe grades of this disease.

Perforated Peptic Ulcer:—The urgency for making a diagnosis in perforated ulcer is usually recognized both by the patient and the physician. It is in this condition, more than with others, that one must not be lulled into inactivity by the relief given by morphine. In the words of Zachery Cope, "Though it may appear cruel, it is really kind to withhold morphine until one is certain whether or not surgical interference is necessary." In recent years, several authors[21,22] have pointed out that perforations may not only occur while patients are being given cortisone, and furthermore, that the symptoms may be modified. This has also been noted after sympathectomy and vagotomy[23]. Although closure of the perforation is urgent, hasty surgery in this condition is not proper as shock and hypovolemia must be combatted with blood and electrolytes. This delay should be utilized for the administration of antibiotics and aspiration of the remaining gastric contents. Conservative methods of management of this condition, with suction and antibiotics, have been evaluated in several centers[24], but on the whole, it still remains a surgical emergency. European surgeons have, for some time, done gastric resections at the time of the emergency surgery for perforated ulcer, and in this country, J. M. Emmett[25] and M. DeBakey have recently presented series of successfully operated cases. In their hands, the original mortality has not been greater than simple closure even though the cases were not selected

in either series. In proper hands and with present day anesthesia, blood and antibiotics, this may prove to be a preferable surgical attack, as it does eliminate secondary surgery and much morbidity.

The subject of perforated ulcer cannot be dismissed without emphasis on spontaneous perforation of the esophagus[26]. Much has been written on this subject in recent years and rightly so, because in the past it has been confused with perforated ulcer, with coronary heart disease and with hydrothorax. Although the condition is infrequent, if it is to be recognized early enough to receive proper curative surgery, it must be even more widely discussed. Sudden high epigastric or precordial pain on straining or vomiting with shock and tenderness brings the patient to the hospital. Along with the film for subdiaphragmatic air which is usually taken for perforated ulcer, the chest should be included as either the mediastinum or pleural cavity may be involved. When the proper diagnosis is made the approach should be transthoracic for suture of the perforation of the esophagus.

On rare occasion, an incarcerated and strangulated diaphragmatic hernia produces an acute abdominal condition unrecognizable except with a chest film. This brings to mind other conditions above the diaphragm causing abdominal symptoms, such as coronary occlusion, pericarditis, and pleurisy.

Comment

This review of acute abdominal conditions has been made to emphasize some of the trends in gastroenterology and surgery in recent years. The old concepts based on careful history and physical and basic surgical principles have not been altered but have been modified to some extent by developments from basic sciences, the laboratory, and special tests. The surgical approach to acute abdominal conditions has been modified by the development and general utilization of gastrointestinal suction, by the use of cortisone and ACTH, by the adequate use of blood, antibiotics, a development of proper methods of replacement of electrolytes and advanced anesthetic technics.

Specifically, the symptoms of appendicitis can be temporarily modified by antibiotics, postponing recognition until perforation has occurred. On the other hand, their control of peritonitis is spectacular and this effect plus aspiration of purulent material at surgery makes possible the removal of the source of the peritonitis even in advanced cases. Admissions for pelvic inflammatory disease are diminishing year by year, but the problem of differentiating appendicitis remains and is just as important as ever. Intestinal obstruction in all its forms has been made safer but its management requires wisdom and diligence in the use of x-ray studies and preparation of the patient. The value of urgent surgery in selected cases of massive gastrointestinal hemorrhage seems established. When all the conditions are right, early surgery in cholecystitis seems advisable but under many conditions, conservative management and interval surgery still seems preferable. Acute pancreatitis is best controlled medically but some

important surgical conditions simulate it closely and even produce repeated high blood amylase levels. The symptoms of perforated ulcer may simulate those of esophageal perforation. Early recognition of the latter and transthoracic approach is essential.

Summary

From a five-year study of hospital discharges, appendicitis, pelvic disease, intestinal obstruction, gastrointestinal bleeding, cholecystitis, pancreatitis and perforated ulcer are the most frequently encountered acute abdominal disturbances. The recognition and management of these conditions are discussed in relation to antibiotics, steroids, blood amylase, transfusions, electrolytes, intestinal suction and new operative technics.

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Discussion

Dr. I. Snapper:—I'm afraid that perhaps not enough appendectomies are done nowadays. We rely for the diagnosis of appendicitis on the same findings as we did thirty years ago, that is, a compatible history and the presence of muscular rigidity in the right lower quarter. Leucocytosis may or may not be present. It is not uncommon to see a child in the evening with some pain in the abdomen but without clear-cut objective signs of appendicitis and still a perforation may take place during the night. Formerly, under such circumstances, the surgeons already went in during the evening, but nowadays we have become so objective that no operation is done on such unclear indication. Perhaps the pendulum has swung too far the other way. In the last ten years I have not seen one case of acute appendicitis in a *physician*, operated before an abscess had formed. In all these cases the diagnosis had been discussed for many days, many x-rays, especially I.V.P.'s had been taken, so that in the meantime an appendicular abscess had developed. It is still better to remove one appendix too many than to overlook one acutely inflamed appendix. With other words, the surgeons have been blamed so often for removing too many appendices that they now have a tendency to operate appendicitis in a late stage when the inflammation has already spread.

It is very interesting to hear that Dr. Snyder has found 1,600 units of amylase in the serum of patients with abdominal diseases other than pancreatitis. This is certainly an important point which will require careful consideration and checking.

Dr. O. H. Wangensteen:—Every one has listened to Dr. Snyder with great attention. I think it is clear that he has had a large experience at the Los Angeles County Hospital in the management of acute abdominal disorders. Some persons, like myself who once had an intimate contact with the problem of abdominal emergencies, and who have become absorbed in aspects of elective abdominal surgery, no longer have the opportunity to see many acute abdominal disorders. A surgeon has to be critical in his appraisal of his own accomplishment to come to have the capacity for judicious analysis of acute abdominal lesions. I am wont to say to a patient or his relatives in the presence of a palpable abdominal tumor, when the diagnosis is in doubt: "If I were to hide an apple or an orange beneath these bed covers, you would have a very difficult time to tell which it is; in fact, it could be a guess. I do not believe you or I should allow the treatment to be guided by a guess which may be only 50 per cent correct".

Dr. Snyder has made an important contribution to the management of intestinal obstruction in infancy and childhood. His record on intussusception is,

I believe, about as good as any reports in the literature. There is one point upon which I would differ with Dr. Snyder. Whereas, I give antibiotics to patients with gangrenous bowel, I confess, I am not as enthusiastic over the protection afforded as Dr. Snyder seems to be. There would appear to be experimental evidence to suggest that in the dog, antibiotics have important value in protecting against the hazards of strangulating obstructions. I have failed to see any evidence of such protection in strangulating obstructions in patients.

Dr. Snyder spoke of strangulated hernia. My erstwhile colleague, Dr. Clarence Dennis of Brooklyn, reported eight consecutive primary resections in infants for nonviable intussusception. In resections for strangulated herniae in adults, Dennis failed to do as well, because old patients in their 8th and 9th decades have other geriatric problems which prejudice a smooth and easy convalescence, as occurs in infants, when careful and timely surgery has been done. The mortality of strangulated hernia in adults in the Dennis series was more than 10 per cent, not because of the ineptness of the surgeon but because of the physical disability which accompanies old age in some of these situations.

In 1889, Charles McBurney of New York, advocated the early excision of the appendix for appendicitis; at the onset of symptoms, McBurney expressed the sanguine hope that early operation would obliterate the mortality of appendicitis. That was a long time ago; however, we are approaching some suggestions of fulfillment of the hope that McBurney expressed more than 60 years ago. The mortality of appendicitis has declined very materially in the past 15 years.

The Metropolitan Life Insurance Company reports a mortality of 1 death per 100,000 policyholders. The vital statistics of the United States indicates a mortality of less than 2 deaths per 100,000 of population. The mortality in the late thirties was 14 deaths per 100,000. In other words, there has been approximately a 90 per cent reduction in the mortality of appendicitis. That is an important achievement. The sharpest decline in mortality occurred moreover before penicillin became available.

Appendicitis is not primarily an inflammatory lesion of the appendix, as its name implies. On the contrary, appendicitis is caused by obstruction to outflow from a small viscus that secretes fluid. As some of you may know, my colleague Dr. Clarence Dennis and I, many years ago examined the appendices of a large number of common laboratory animals as well as the appendices of a host of other animals not commonly used in the experimental laboratory. Dennis found only three animals in which the appendix secreted fluid. One was the rabbit; the second was the chimpanzee, and the third was man. All the others, including most any kind of animal, zoo or otherwise, had appendices which did not secrete fluid when its appendix was obstructed.

In the rabbit, chimpanzee and man intraluminal pressures, in the presence of obstruction of the base of the appendix, were found in the area of 100 mm. of Hg—a pressure in excess of diastolic blood pressure. Long ago, Starling found that the parotid gland in the dog will secrete against a pressure of 250 mm.

of Hg. No organs of the body have a physiological secretory pressure as high as we observed in the appendix of the rabbit, chimpanzee and man. It is understandable, therefore, that when the appendix is obstructed that, a chain of damaging events is set in motion that will threaten the viability of the appendical wall: leucocytic infiltration and softening of the wall occur in consequence of a mounting pressure which first exceeds venous pressure, then successively capillary arteriolar and diastolic pressure. Presently, the bursting strength of the appendical wall has been lessened to the extent that, perforation will occur at a very low pressure. Antibiotics do not lessen the great secretory capacity of the rabbit's appendix. Antibiotics can keep the hazard of perforation temporarily in check. The treatment of appendicitis today, however, as in McBurney's day, is *early* excision of the appendix at the *very* beginning of symptoms.

Surgeons have talked a lot about unnecessary surgery. I am sensitive over unnecessary surgery. I do not believe in willy-nilly appendectomy as an isolated operation. A few surgeons apparently do. I know of a few instances in which surgeons arranged for the excision of the appendices of their children in the absence of symptoms. When I am in the abdomen, however, I frequently remove the appendix if it looks normal, because of the potential hazard of future obstruction of the appendix. I would say, it is permissible to remove the appendix on *slight* but *definite* indication. One certainly should not wait until the symptoms of rupture are in evidence. Unnecessary appendectomy is probably not a frequent operation.

I would put fixation of the uterus at the head of the list of unnecessary operations performed currently in this country. If any surgeon in this room suspends uteri, I would say you are probably doing unnecessary surgery. Indications for suspending the uterus, must be very few indeed. High on the list of unnecessary operations, too, should be hemorrhoidectomy. To be certain, there are satisfactory and acceptable indications for doing the operation. Persistent bleeding and prolapse are such indications. I see a good many patients in the cancer age. Proctoscopy and sigmoidoscopy are regular routine diagnostic procedures for such patients. They are done by full-fledged proctologists; yet, of the patients they advise to have hemorrhoidectomy, I suggest to about 5 per cent of them that, they accept the advice and have the operation done. The other 95 per cent do not need hemorrhoidectomy, in my opinion. Everyone has hemorrhoids. What justification is there for willy-nilly hemorrhoidectomy? It is important to exclude cancer by a careful diagnostic visual examination carried out by a trained proctologist. But there is no justification for the frequency with which hemorrhoidectomy is done today in many hospitals. Look at the schedule of posted operations in the operating room suite of many voluntary hospitals! Uterine suspensions and hemorrhoidectomy, without adequate and acceptable indication, are operations which will not appear as frequently on those schedules presently, when surgeons and patients alike come to know that these operations rank high on the list of "unnecessary operations".

GASTRIC CANCER, WITH A CONSIDERATION OF TOTAL GASTRECTOMY*

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The noted German poet, Theodore Storm, died of gastric cancer. He wrote a poem entitled *"Beginn des Endes"* concerning his illness, which described poignantly the insidious onset of gastric cancer. The poem is as follows[1]:

'Tis but a prick, 'tis scarce a pain,
Just felt, to which no name you give:
Henceforth it speaks again—again,
Uneasy now you have to live.

If to complain you try—of what?
You cannot put it into speech:
Within you say, "Indeed 'tis naught!"
Henceforth it holds fast like a leech.

So seldom strange your world does grow,
And quickly are you stript of hope,
Until at last you really know
That with Death's shaft you cannot cope.

Storm died in 1888, seven years after Billroth had first operated successfully for gastric cancer.

Pessimism exists in the minds of many physicians in their regard for the surgical treatment of gastric cancer. This pessimism has led to a fatalistic view in the minds of these same physicians and has resulted in a lack of enthusiasm for recommending operative treatment. Statistics gathered throughout this country and abroad suggest that in the past only between 5 and 10 per cent of all persons coming to clinics and hospitals, suffering from gastric cancer, survived for five years or more. This figure includes all of those patients suffering from gastric cancer, whether inoperable or whether the operation be performed simply as a palliative maneuver. If, however, one excludes such palliative operations and the inoperable cases, a better prospect comes into view.

For example, Balfour, writing in 1937[2], stated that when the growth and regional glands accompanying the gastric neoplasm can be extirpated, 30 per cent of these patients will live for five years. Furthermore, when there are no nodes involved, a situation which obtains about one-fourth of the time, nearly half will live for five years; and in the presence of positive nodal involvement, one-fifth of the patients will be alive and well at the end of five years. In the ensuing 15 years, an improvement in operative mortality, coupled with an increased willingness to explore all lesions of the stomach surgically, sheds a

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new hope for the 40,000 persons who will die from gastric cancer in the United States this year.

Gastric cancer is curable. The problem at the present day is to recognize the disease and direct the patient into proper channels where *adequate* and skillful surgical treatment will be carried out expeditiously.

In regard to the incidence of cancer of the stomach, Livingston and Pack[3] have written as follows: "There are more deaths from cancer of the stomach than all malignant tumors of lip, tongue, cheek, tonsil, pharynx, larynx, salivary glands, thyroid, male and female breast, ovary, uterine cervix and corpus uteri *combined.*"

The incidence of cancer of the stomach as reported in the registry of the California Hospital Tumor Clinic in Los Angeles is approximately 1 in 10 patients, and in charity hospitals the incidence is higher. In a summary of the experiences of 9 hospitals in Califoria between 1942 and 1948[4], the California Tumor Registry reported a total of 5,133 neoplasms involving the digestive organs. Approximately one-third of these lesions implicated the stomach. The total number of neoplasms encompassing all parts of the body, reported during this period, was 20,511. The incidence of cancer of the stomach in this series was slightly lower than cancer of the female breast, cervix uteri and skin.

Further evidence that there is room for improvement in the future exists in the statistical study carried out at the University of Minnesota[5]. Of the 14 patients who were operated upon in 1944 with negative node involvement, 71 per cent survived for three years. There was a 64 per cent survival for five years. In addition, of 67 patients who were operated upon in a five-year period (with negative nodes), 63 per cent survived for three years and 51 per cent were alive for five years. In this same five-year period, 43 per cent of all patients who were operated upon for cure, with or without nodal involvement, had survived for three years, and 33 per cent of all these patients were alive and well at five years.

These figures are in contrast with the general statistical analyses, indicating that only between 5 and 10 per cent of all persons entering hospitals with gastric cancer survive for five years or more.

The Problem of Gastric Ulcer

The excellent analysis of Banks and Zetzel[6] emphasizes the shortcomings of the medical management of patients with gastric ulcer. Forty-eight cases of gastric ulcer selected for medical management were reviewed. Four patients were followed for more than 15 years; 7 for 10-15 years; 20 for 5-10 years; and the remainder for an average of two and a half years. The commonly accepted criteria for differentiating the benign and the malignant ulcer—"honored more often in the lecture room than at the bedside"—included prompt subsidence of ulcer symptoms under treatment; presence of free hydrochloric acid in the

stomach and consistent absence of occult blood from the stools; satisfactory gain in weight; complete disappearance of the ulcer niche on roentgenologic observation within six to eight weeks and full return of pliability in the gastric wall at the involved site. In selected cases, the information derived from gastroscopic study and from the staining of gastric washings was utilized.

Of the 48 patients, only 11 remained free of ulcer symptoms during the follow-up. An additional 7 have remained well after one or more recurrences of ulcer activity. Twenty patients continued to have periodic exacerbations of ulcer symptoms. Six of these eventually submitted to operation; 4 others similarly advised refused operation. *Eight patients of the original group of 48 have died of verified carcinoma of the stomach.*

It is generally believed that from 10 to 20 per cent of patients who are operated upon for an ulcer presumed to be benign by clinical criteria are found to have a carcinomatous lesion[7,8]. In the group of preferred risk patients studied by Banks and Zetzel, only 1 of 4 patients remained symptom free over the years; 1 of 6 died of carcinoma of the stomach; and 1 of 5 was operated upon or refused operation despite urgent indications. It is probable that with the passing of time other patients in this group will be forced to accept operation, and some may develop gastric carcinoma.

Indeed, a physician assumes grave responsibility for the future welfare of his patient when a program of conservative therapy is elected for an ulcerating lesion of the stomach presumed by usual criteria to be benign. The prognosis of gastric ulcer under medical treatment, as reflected in the probability of present or future carcinoma and in the incidence of recurrences and complications, is far from reassuring. A similar group of gastric ulcers treated by routine gastrectomy and followed for a comparable length of time should show certain accrued statistical benefits over the group treated medically. This benefit can, of course, accrue only where the gastrectomy is carried out in experienced hands with a negligible mortality rate. It is well known that gastrectomy for gastric ulcer is one of the most gratifying of all operations for lesions of the upper gastrointestinal tract. The morbidity and mortality are lower than for either duodenal ulcer or gastric carcinoma, and the technical features of the operation are infinitely easier. Fifty consecutive gastrectomies for gastric ulcer without a mortality is not a figure in the realm of speculation. It appears that there is need for wider publicity of the known facts concerning gastric ulcer, both among the profession and the public.

The important thing is that there is no incontestable means of determining whether a given ulcer is benign or malignant except by microscopic examination. It is idle and ridiculous to speculate upon what a gastric ulcer may have been yesterday or will become tomorrow. Therefore, all must be viewed with suspicion. Discussion of what may transpire at some indefinite future date leads to indecision and procrastination. About one-third of all gastric cancers are of the ulcerating variety, including those which ulcerate secondarily. Consequently,

the prompt resection of gastric ulcers would not solve the problem of early diagnosis in gastric cancer entirely.

Adenomatous Polyps

The infrequency of gastric polyps, when contrasted with gastric cancer, suggests that these benign lesions are responsible as precursors for only a few of the clinical gastric cancers. Spriggs[9] estimated the incidence of malignancy

Fig. 1—An inadequate subtotal gastric resection performed for an ulcerating carcinoma of the lesser curvature. The proximal line of resection is dangerously close to the tumor, and there is a striking absence of greater, gastrocolic, gastrohepatic and gastrolineal omenta. This type of operation may, in part, be responsible for the poor results obtained in the surgical treatment of this disease in the past.

in polypoid disease of the stomach from 18 to 28 per cent. Benign polyps are likely to be associated with reduced gastric acidity or even with achlorhydria. In a group observed by Cromer, Comfort and Butt[10], the mean free acidity was 16° lower and achlorhydria 65 per cent more common than the normal for persons of the same age and sex. Certainly gastric polyps, when recognized, should be removed promptly.

Primary Pernicious Anemia

Rigler and Kaplan[11] are mainly responsible for the concept that primary pernicious anemia is a precursor of gastric malignancy. They reviewed a part of the autopsy material at the University of Minnesota Hospitals and a selected group with an established antemortem diagnosis of pernicious anemia. They found that 7 per cent were associated with gastric cancer. Others have felt this figure to be somewhat high, but there seems little doubt that a correlation exists between the presence of primary pernicious anemia and the likelihood of developing gastric carcinoma.

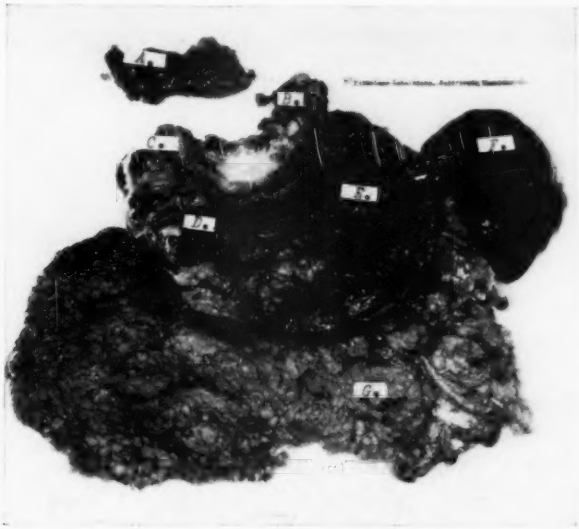

Fig. 2—A. Pancreas. B. Lymph node. C. Duodenum. D. Tumor. E. Stomach. F. Spleen. G. Greater omentum.

A "radical" total gastrectomy with excision of all important lymph gland-bearing structures. Removal of the spleen and the tail of the pancreas in this instance resulted in extirpation of involved lymph nodes, which would have been overlooked otherwise. There is a generous excision of the proximal duodenum. Total gastrectomy was necessitated by the presence of infiltration along the lesser curvature proximally.

Lesions in the lower half of the stomach can be treated by this same radical principle with preservation of a small proximal gastric stump as long as the principles outlined above are observed.

Achlorhydria

Ivy[12] states that 35 per cent of the people over the age of 60 do not secrete acid after a test meal, and that in 28 per cent there is no response even to histamine. Inasmuch as achlorhydria is found in some but by no means in all cases of gastric cancer, this test alone offers little in the early detection of cancer of the stomach. Many authors regard the presence of achlorhydria as an associated rather than a causative phenomenon in the presence of gastric cancer.

Consideration of Total Gastrectomy

The poor results which have been attributed generally to gastrectomy in the treatment of cancer in the past may, in part, be attributed to certain omissions in the technical features of the operation. The conservative gastrectomy of 5-10 years ago as performed by a large number of surgeons is now regarded as obsolete as simple mastectomy for the eradication of cancer of the breast. A surgeon, for example, who today fails to remove the spleen for a cancer of the body of the stomach ignores the fact that betwen 20 and 47 per cent of cancers of the stomach show metastases to the splenic pedicle. In the past, total gastrectomy has been reserved for those malignant lesions of the stomach without distant metastases which could not be removed locally by subtotal resection. Figure 1 illustrates an inadequate sleeve type of resection employed for an

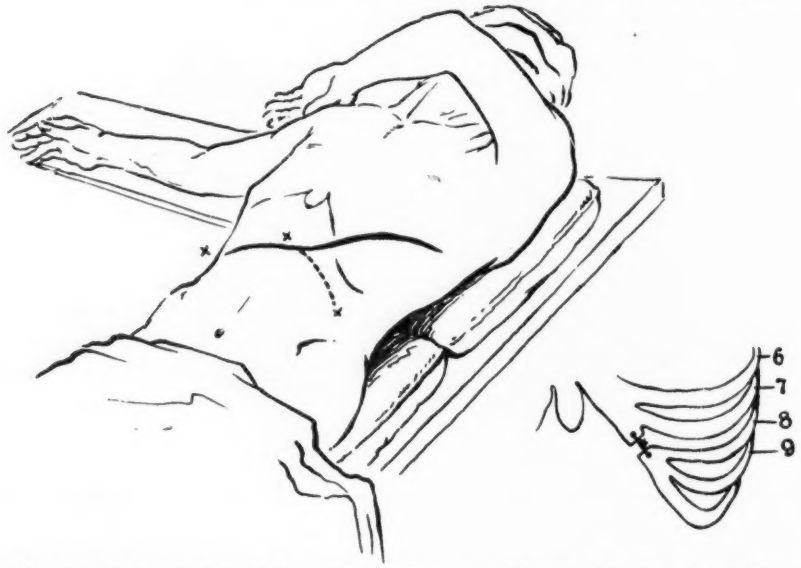

Fig. 3—Illustrates the position of the patient upon a rotational table for gastrectomy. In the event that the thoracoabdominal incision is used, the patient is rotated to the right.

ulcerating gastric neoplasm. The fundamental criteria in the treatment of cancer have not been fulfilled; namely, excision of the primary tumor and associated lymph gland-bearing structures. The absence in this specimen of the greater, gastrohepatic and gastrocolic omenta as well as the gastrolineal structure is in contrast to the specimen illustrated in Figure 2.

One or two technical features are perhaps worthy of comment, applicable to both radical subtotal gastrectomy and total gastrectomy. Figure 3 illustrates the position of the patient upon a rotational table. The right half of the transverse incision is performed first and an abdominal exploration then carried out. If the lesion appears to be limited to the lower end of the stomach, the incision is simply extended to the left along the course of the dotted lines, as illustrated

in Figure 3. If the lesion extends into the proximal portion of the stomach and it is apparent that total gastrectomy will be necessary, the incision is then extended conveniently across the costal arch and into the seventh intercostal space up to the anterior axillary line. The diaphragm is also opened widely, allowing excellent exposure. It is our custom to resect a portion of the costal cartilage where it is divided so that there will not be pain due to rubbing of the apposed incised surfaces (See inset Fig. 3).

Figure 4 illustrates the exposure from this type of incision. The use of long stay sutures with apposition of the jejunum to the peritoneal covering of the

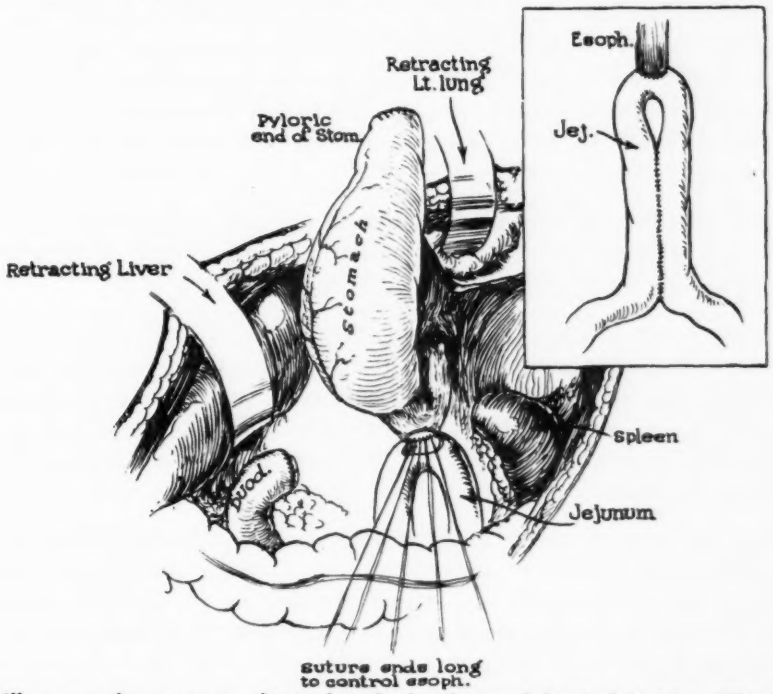

Fig. 4—Illustrates the exposure obtained with the thoracoabdominal incision. The long stay sutures are applied to the esophagus before it is divided. Completion of the posterior row of the anastomosis is done with the stomach *in situ*. Traction upon the stomach provides excellent exposure of the anastomotic site. Inset illustrates the long enteroenterostomy.

diaphragm facilitates the anastomosis. Also, a long 6 or 8 inch enteroenterostomy seems to aid in the postoperative management of digestion. These loops apposed provide a double lumen which serves as a substitute for the reservoir function of the stomach.

The high mortality of total gastrectomy, together with the uncertainties of digestion and metabolism in the absence of the stomach, has restricted the use of this operation in the treatment of cancer. Longmire[13] pointed out in 1947 that if these objections could be obviated, undoubtedly block excision of the

stomach and regional lymph nodes should be the most effective means of treating neoplasms of the stomach. This philosophy is particularly logical when one compares the conventional subtotal gastrectomy with the operations generally accepted for cancer of the breast and rectum. At that time, he reported a series of 20 total gastrectomies with 2 operative deaths. When one reviews the lymphatic drainage of the stomach critically, it becomes evident that the operation of subtotal gastrectomy fulfills the major premise for any operation for malignancy inadequately; that is, the ability of the procedure to remove the lesion together with the adjacent lymph gland-bearing structures.

The mortality of 133 total gastrectomies, as reported by Lahey[14], is as low as, if not lower than, the mortality of subtotal gastrectomy. Of these total gastrectomies from 1944 to October, 1949, there were 58 cases with 2 deaths, or a mortality of 10.3 per cent. From 1947 to 1949, 36 total gastrectomies were performed with 3 deaths, or a mortality of 8.3 per cent. Partial gastrectomy, on the other hand, from 1947 to 1949 was accompanied by a mortality of 9.2 per cent.

In a personal series[15,16], 29 total gastrectomies have been performed, with 2 deaths. One of these operations was carried out in a patient at the age of 89; one 82 and one at 79. There were no deaths in this older group.

The three-year survival rate of total gastrectomy in Lahey's[14] series was as high as the average five-year survival rate for carcinoma of the stomach treated by subtotal gastrectomy. This is of significance when one appreciates that up to then total gastrectomy had been reserved only for those patients with far advanced, and frequently what were thought to be hopeless, lesions involving almost all of the stomach.

It does not appear, however, that this is the time to propose that everyone who operates upon a patient for cancer of the stomach should carry out total gastrectomy, but it seems certain that the operation of subtotal gastrectomy should be revised in the direction of radicalism so as to include lymph gland-bearing structures.

Analysis of the problem can be carried out accurately only through careful pathologic studies. Such a study has been reported by Eker[17], who conducted meticulous gross and microscopic pathologic investigations of the specimens from 70 total gastrectomies and 100 partially resected stomachs. The incidence of involved lymph nodes was 70 per cent in the subtotal and 82.9 per cent in the total gastrectomy group. The differential in incidence of nodal involvement existed in the region of the lesser curvature, suggesting that the defection of subtotal gastrectomy exists along the celiac axis and the gastrohepatic omentum rather than in linear dimension. Thirty-two per cent of all the gastric carcinomas, either in the totally or subtotally resected group, when involving the upper part of the stomach, showed microscopic involvement of the esophagus. This fact would support the view that total gastrectomy is mandatory when the lesion, regardless of size, is limited to the upper half of the stomach. In 19 annular car-

cinomas, 9 showed metastases to the splenic pedicle; an incidence of 47.4 per cent. In 46 random specimens where the spleen was removed, the over-all incidence of involvement of the splenic pedicle was 21.7 per cent. Failure to remove the spleen, whether total or subtotal gastrectomy is carried out, will, therefore, relegate a significant number of patients to doom. The average number of nodes was 22.6 per specimen in the total group and 12.6 nodes per specimen in the subtotal group, suggesting that the extirpation of lymph nodes can be carried out more adequately by total gastrectomy.

Templeton, et al[18], on the other hand, analyzed 18 total gastrectomies. Only 5 of these patients survived for a period of six to 51 months after operation. There was one operative death. Of the 12 who lived, the average length of life was 11 months. They concluded from their experience that there is little evidence to support total gastrectomy as a means of improving the results in the treatment of cancer of the stomach.

Pack and McNeer[19] have also stated that they feel total gastrectomy is only indicated for those tumors where subtotal gastrectomy would be ineffectual for gross removal.

In a series of 453 resections for gastric cancer, Harvey, et al[20] reported that almost 30 per cent of these specimens showed no evidence of lymphatic metastases, indicating that total gastrectomy in this group at least would be unnecessary, and if the mortality rate were increased significantly, theoretic benefit accruing to the other 70 per cent would be negated. In a further attempt to evaluate the role of total gastrectomy in the treatment of cancer of the stomach, 270 cases of resection were studied carefully for evidence of invasion of neighboring viscera. None was found in 93. There was extension to the duodenum in 53, omentum 77, esophagus 37, and diaphragm 6. In 210 patients who suffered recurrence, only 21 showed clear evidence of carcinoma persisting in the gastric remnant after satisfactory subtotal gastrectomy. All of these patients also showed distant metastases which were possibly seeded before the original operative maneuver. This observation suggests that in only a few patients is linear dimension as important a consideration as excision of the spleen and adjacent omenta. In 3 of 60 survivors, tumor was thought to be present in the resected margin at the time of pathologic examination. *All 3 of these patients, however, lived for 10 years!* In an additional 18 patients who survived, the plane of excision was less than 2 cm. from the tumor, and in only two-thirds of these survivors did a wide excision pertain.

Dunphy[21] has stated: "Consistently similar results of well performed surgery of the breast, stomach, colon and rectum over the years in the hands of different surgeons (for example, 7 to 10 per cent five-year survival for cancer of the stomach, 50 per cent survival for cancer of the breast, and 45 per cent survival for cancer of the colon) suggests that this represents about the number of tumors biologically susceptible to surgical extirpation. It seems unlikely that

superradical excision as a routine will materially affect these figures, but in the hands of all but the most gifted surgeons it will significantly increase mortality and morbidity."

He states that total gastrectomy with resection of the pancreas, spleen, colon, adjacent liver, and all intervening lymphatic tissues is an acceptable operation on the other hand for a huge, invasive, apparently slowly growing carcinoma of the stomach that has invaded locally without distant spread. He feels, however, that this procedure as a routine for cancer of the stmoach is unthinkable. He raises the following objections to the routine use of total gastrectomy for all operative gastric carcinomas. First of all, it is evident that total gastrectomy is not always necessary. Secondly, it is unquestionably a more disabling procedure to the patient than a radical subtotal gastrectomy. Thirdly, it is not a good palliative operation, being tolerated particularly badly by older patients with recurrent disease. In the over-all picture, it will increase the morbidity and mortality of gastric surgery for cancer, neither of which can be overlooked. Finally, it will not solve the problem of the very poor results in gastric carcinoma. It adds to the total survival period chiefly when it is used for a slowly growing, biologically favorable tumor that cannot be encompassed by subtotal gastrectomy. The incidence of such cases is low, but they constitute a brilliant example of the proper application of the more radical operations.

McNeer, et al[22] in a critique of subtotal gastrectomy, pointed out that 34.1 per cent of those remaining free of disease were apparently cured by subtotal gastrectomy. A review of the postmortem protocols in 92 cases of subtotal resection was carried out. The recurrence rate in the gastric remnant was 50 per cent; in the duodenal stump 14 per cent. Metastases to perigastric lymph nodes were apparent in 22 per cent of the cases. In other words, in approximately 80 per cent of the instances, subtotal gastrectomies had failed in the local eradication of the disease. These studies suggest that improvement could be expected from removal of more stomach and more duodenum. Eight of 15 carefully studied specimens in their series showed metastases to the splenic pedicle or to the inferior or superior border of the pancreas, suggesting that the spleen and tail of the pancreas must be removed in an adequate gastrectomy, whether it be total or subtotal. Four of 6 patients with multiple organ resection prior to 1943 remained well for five years without recurrence. This is of particular significance because at that time, these operations were done only for apparently far advanced or well nigh hopeless disease. This also supports the rationale for applying this principle to lesions of lesser extent.

McNeer, et al[23] conclude from a careful evaluation of the problem that subtotal gastrectomy, as commonly practiced in the past, may have denied the chance for permanent survival to about 50 per cent. Because of development of recurrence in the gastric remnant, duodenal stump, perigastric lymph nodes or stomach bed, they suggest two courses:

a. Standardize the radical subtotal gastrectomy; or

b. Employ total gastrectomy routinely for all operable cancers of the stomach.

Thomas, Waugh and Dockerty[24], in attempting to evaluate this problem, suggest that the rationale of carrying out total gastrectomy for all cancers of the stomach, regardless of size, might be analogous to removing the entire face for a small basal cell carcinoma. They mention the widespread opinion that recurrence in gastric or duodenal stump is rare. They also suggest that the results of the series reported by Scott and Longmire in 1945 are disappointing inasmuch as only 18 per cent of their patients were alive one year or longer after operation, indicating that there is little difference between what can be expected from total or subtotal gastrectomy. They also imply that improper total gastrectomy may well be less effective in the eradication of cancer of the stomach than a properly performed subtotal gastrectomy.

It is not time to propose that everyone who operates upon a patient for cancer of the stomach should carry out total gastrectomy, but it is perhaps time to revise some of our thoughts concerning the operative maneuver carried out for the treatment of this disease.

Summary and Conclusions

1. It would appear that, contrary to widespread opinion, recurrence at the proximal site of resection is more common than generally appreciated, and every effort should be made to extend the line of resection well beyond the limits of the tumor. In lesions of the upper portions of the stomach, this will necessitate total gastrectomy. In lesions of the lower half of the stomach, such should not obtain necessarily.

2. Recurrences in the region of the duodenal stump are, likewise, more common than generally acknowledged, and every effort should be made to excise a generous portion of this structure. Whether total or subtotal gastrectomy is carried out will not influence this particular extension of the operation.

3. A significant percentage of cancers of the stomach involves the gastrolineal ligament and, therefore, necessitates removal of the spleen. Lesions located in the midportion of the stomach with macroscopic annular infiltration may show metastases to the splenic pedicle—an incidence as high as 47 per cent[17]. The importance of removing the spleen probably far outweighs the necessity for carrying the extent of the resection longitudinally into the esophagus.

4. A significant number of patients with cancer of the stomach shows involvement of the omentum. An excision of the greater omentum as well as the gastrocolic and gastrohepatic omenta should be employed as a routine in either total or subtotal gastrectomy.

5. Involvement of the group of lymph nodes along the superior and inferior borders of the tail of the pancreas is also quite common, and suggests that routine excision of the tail of the pancreas distal to the origin of the splenic vein might be a natural accompaniment of either subtotal or total gastrectomy. Multiple resection of viscera, including transverse colon, lobes of the liver, diaphragm and left kidney, may result in worthwhile salvage in certain instances. Anyone operating for a carcinoma of the stomach should be prepared to carry out such a maneuver in the event that it is indicated.

6. Total gastrectomy should not be performed for palliation.

7. Total gastrectomy is probably not necessary in low-lying lesions of the antrum as long as the principles outlined above are observed.

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Discussion

Dr. O. H. Wangensteen:—I listened with a great deal of interest and approval to Dr. Farris' very nice paper. He omitted to say that he himself was the author of a very nice paper on total gastrectomy (1943). In his paper of 10 years ago, Dr. Farris spoke of the use of iron in combating the anemia of gastrectomy. For years, I followed Dr. Farris' advice of giving iron to such patients.

One of my associates, Dr. Fred Cross, has shown very nicely that the esophagus is very susceptible to injury by bile and pancreatic juice. After total gastrectomy, by the end-to-side technic, even though an enteroanastomosis between the afferent and efferent jejunal loops is made, an erosive esophagitis develops which causes a macrocytic anemia. Ultimately of course, all total gastrectomized patients do develop a megaloblastic anemia which requires the para-oral administration of Vitamin B_{12}. If a Roux-Y anastomosis is made, the macrocytic iron deficient anemia can be avoided, for erosive esophagitis does not follow this procedure.

Erosive jejunitis or ileitis with anemia occurs remotely after side-to-side anastomoses following intestinal resections. I have seen 3 such cases. It is, of course, a hydrostatic phenomenon. End-to-end anastomoses avoid this occurrence.

Dr. Farris spoke of a patient of 89 and of another patient of 82 and another of 79 years—all of whom survived excision of an esophageal diverticulum. I do not know of any surgeon who has operated on a hundred patients of these ages who had a diverticulum of the esophagus. As I said yesterday, I believe one could do gastric resection for patients in their early eighties who were in reasonably good physical condition at a risk not exceeding 15 per cent.

I would subscribe to everything that Dr. Farris said concerning amplification of gastrectomy for cancer. Since I have been here, I have had called to mind the paper by Drs. Coller, Kay and McIntyre in the *Archives of Surgery* for 1941. They stressed the great importance of adequate lymph node removal in the operation. Every surgeon would do well to study their diagram of the broad extent of the location of the regional lymph nodes of the stomach. The spleen and its pedicle should be excised regularly. This circumstance demands

at least an 85 per cent excision. That means the surgeon must have ready access to the attic of the abdomen. Even for a pyloric cancer, it is necessary to do an 85 per cent resection to be certain that all the lymphatic drainage area is excised.

For more than 4 years in our clinic, we have had a fairly well standardized operation for gastric cancer: 85 per cent of the stomach is excised regularly; total gastrectomy together with excision of 5 cm. of esophagus and 4 cm. of the duodenum is done, if the margin of normal gastric tissue at the upper end is close to the tumor; the hepatic artery and celiac axis are cleared of lymphatic bearing tissue; the superior border of the pancreas is cleared, as are the crura of the diaphragm in juxtaposition to the esophagus. If the lymph nodes along the superior border of the pancreas are involved, the portion of the pancreas, distal to the site of involved lymph nodes, is removed. The gastrohepatic omentum is removed as is the entire greater omentum; the *vasa breviae* which enter the posterior wall of the stomach ordinarily suffice to nourish a 10 to 15 per cent gastric fragment. If in the excision of the splenic pedicle, these should be damaged, or if partial pancreatectomy is done together with excision of the main trunk of the splenic artery, total gastrectomy, of necessity, must be done.

Surgeons, in recent years, have been emphasizing the importance of extending the upper line of resection higher and higher. Some surgeons affect to believe that total gastrectomy should be done regularly for gastric cancer. The completeness of the lymphatic drainage area is equally important. In the subpyloric area, along the greater curvature, surgeons probably have not been as thorough in the removal of lymph nodes as the situation warrants. There is no "pyloric block" at the pylorus. A good length of duodenum should be excised regularly. Yes,.I have done it on several occasions to within a few millimeters of the termination of the biliary ampulla. Obviously, a probe must be put into the common bile duct during such dissections.

My colleague, Dr. Stuart Arhelger, in studying the sites of observed residual cancer in "second look" operations for gastric cancer, tells me that there appear to be 2 weaknesses in our currently practiced operation, which most of us have looked upon as quite radical. Arhelger found residual cancer most frequently at 2 sites: along the superior border of the pancreas and along the component structures contained within the right extremity of the gastrohepatic omentum as the bile ducts, portal vein and hepatic artery enter the *porta hepatis*. Since this analysis of the findings in the "second look" procedures for gastric cancer, we have added dissection of the triangle between the gallbladder and the common bile duct structures as a part of the primary procedure. This dissection occasionally makes it mandatory to excise the gallbladder or to drain it. Moreover, we are now excising a goodly portion of the pancreas in the presence of suspicious lymph nodes along its superior border, on less indication than previously.

MALIGNANT DEGENERATION IN ATROPHY OF THE STOMACH AND PERNICIOUS ANEMIA*

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and

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Numerous articles have been written in recent years concerning the relationship between carcinoma of the stomach and the atrophy of the gastric mucosa which regularly accompanies pernicious anemia as well as that which may occur independently. In order to determine the incidence of carcinoma of the stomach in patients with atrophic gastric mucosa, a series of 110 patients, all of whom exhibited this change, was examined. All were patients at the Los Angeles County General Hospital between the years 1942 and 1952. Eighty-five had an established diagnosis of pernicious anemia, while the remaining 25 exhibited only

TABLE I

AGE, RACE, AND SEX INCIDENCE

Decades		2	3	4	5	6	7	8	9	Total
Caucasian	Male	1	1	3	7	11	25	8	1	57
	Female	–	2	2	3	6	23	11	1	48
Negro	Male	–	–	1	–	3	1	–	–	5
	Female	–	–	–	–	–	–	–	–	–

Total Cases 110

the gastric mucosal atrophy. While the diagnosis of pernicious anemia was established by standard methods, that of atrophy was established solely by gastroscopy or microscopic examination of autopsy material. It should be noted that the most marked atrophy consistently occurred in the cases of pernicious anemia.

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Careful study of the history of each of the 110 patients from the time of first admission to final outcome yielded three cases of gastric malignancy. In one of these, a histologic diagnosis of plasma cell cytoma was established following gastric resection, while in the second case, the autopsy diagnosis was leiomyosarcoma. In the third patient, a roentgenographic diagnosis of gastric neoplasm was made in an individual with palpable abdominal masses.

TABLE II

SUMMARY OF FINDINGS IN 110 CASES OF PERNICIOUS ANEMIA AND ATROPHY OF THE STOMACH

Pernicious Anemia		85
Atrophy		25
Free acid	No free hydrochloric acid	45
	Study not done	62
	Free hydrochloric acid	3
Upper G.I. x-ray	Study done	47
	Study not done	63
Gastroscopy	Done	58
	Not done	52
Years of proved pathology	0–1	2
	1–5	40
	5–10	7
	10–20	18
	20–	5
Autopsy done		8
Death–No autopsy		11
Malignant neoplasms		3
Benign neoplasms		5
Associated gastritis	Superficial	1
	Hypertrophic	1

DISCUSSION

Ninety per cent of the patients studied (Table I) were beyond the fourth decade, with the youngest patient aged 18 and the oldest 93. A total of five Negro patients (all males) occurred in the entire group, and of these, four had pernicious anemia. The series includes 62 males and 48 females.

Our investigations confirm the consistent coexistence of achlorhydria and gastric mucosal atrophy (Table II). Only three of 48 patients studied showed

the presence of even traces of free hydrochloride and none of these had pernicious anemia. Upper gastrointestinal series, done in 47 patients, yielded no abnormalities with the notable exception of the one patient, referred to previously, in whom a roentgenographic diagnosis of gastric neoplasm was established. Gastroscopy confirmed the presence of gastric mucosal atrophy in each of 58 patients, while autopsy revealed atrophy in eight more. Of those gastroscoped, superficial gastritis occurred in one patient and hypertrophic gastritis was a coincidental lesion in another. Seventy-two patients with pernicious anemia were under observation for periods ranging from a few months to more than 20 years. Thirty of this number remained under observation from five to more than 20 years. In addition to the eight autopsied patients already alluded to, death occurred in 11 patients, in none of whom was there clinical suspicion of malignancy. Benign neoplasia, as exhibited by the presence of gastric polyps, was demonstrated by either gastroscopy or x-ray in five patients.

It may be well to discuss in some detail the three patients in whom malignant neoplasms occurred. In the first of these, a 69-year old Negro male, the diagnosis of pernicious anemia with combined system disease was well established. An upper gastrointestinal series demonstrated a polyp of the greater curvature and the gastroscopist (D. N.) described a malignant polypoid mass in addition to marked gastric mucosal atrophy. Surgery disclosed a polypoid mass on the greater curvature which, histologically, proved to be a plasma cell cytoma. Our second patient, an 86-year old Caucasian female, had had pernicious anemia for 24 years. The presence of palpable abdominal masses led to an upper gastrointestinal series which revealed several large filling defects in the stomach. Permission for autopsy was denied. The third patient, a 79-year old Caucasian female had been treated for pernicious anemia for three years. Death followed massive gastrointestinal hemorrhage, the source of which was found at autopsy to be a gastric neoplasm. The neoplasm was a 3 cm. rounded submucosal mass near the pylorus. Histologically, the diagnosis of leiomyosarcoma with ulceration was established.

Comment

The general medical literature reveals an incidence of three per cent to ten per cent for gastric carcinoma developing in patients with pernicious anemia. Mosbech and Videbaek[1], in studying 301 patients with pernicious anemia, found that the likelihood of gastric cancer occurring in this condition is three times the expected incidence in the general population. While our incidence of 3.5 per cent for malignant neoplasm corresponds rather well to these figures, it should be stressed that no proven case of actual *gastric carcinoma* occurred in our series. In a survey of more than one thousand cases of gastric carcinoma, LaDue, Murison, McNeer, and Pack[2] found a single instance of this lesion developing in a patient with pre-existing pernicious anemia.

SUMMARY

1. One hundred and ten patients with gastric mucosal atrophy have been studied.

2. Eighty-five of these patients had an established diagnosis of pernicious anemia.

3. In the pernicious anemia group, three patients (3.5 per cent) developed malignant neoplasms of the stomach, but none of these was definitely proven to be carcinoma.

4. In the 25 patients with gastric mucosal atrophy only, no case of malignant gastric disease was encountered.

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PERITONEOSCOPY AS A DIAGNOSTIC ADJUNCT IN GASTROENTEROLOGY*

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Peritoneoscopy, the visualization of the abdominal cavity by means of an optical instrument, is a most useful diagnostic adjunct in the differential diagnosis of abdominal disease. It is not the purpose of this paper to make any plea for the procedure, it needs none. The value has been proven over the past 21 years and can readily be demonstrated to any unbiased physician with one or two cases done by the experienced peritoneoscopist. I will attempt to show you some of the facets of peritoneoscopy that are most valuable as an aid in gastroenterology, with some illustrative cases. Peritoneoscopy was not designed to supplant laparotomy; but in many cases it can answer a diagnostic problem, as well, and often better than the purely exploratory laparotomy. Cases should be selected according to criteria previously described and with a definite purpose in mind. When this purpose is accomplished then the procedure has been a success. Negative information is often as valuable as positive findings.

While peritoneoscopy had been done in Europe since 1910, only a very few cases had been done in this country up to 1924. The procedure seemed to be dropped about then for want of adequate instruments and a safe technic. In 1932, Dr. John Ruddock of Los Angeles and I started working on the problem and, by 1934, the basic instruments of the Ruddock scope were evolved and Dr. Ruddock published his first paper. By 1937 the present instruments were available and a safe, simple technic worked out. This lead to a renewed interest in the procedure and it is now widely carried out throughout this country.

A brief resumé of the technic of peritoneoscopy is now in order. It should be carried out under aseptic conditions in an operating room that can be darkened, on a table that will tilt not only in the Trendelenburg position; but from side to side. The patient should be prepared by an enema the night before; the stomach and bladder should be empty. Preoperative medication is given according to the age, weight and general condition of the patient. We are careful not to over medicate since so many of the patients we see are elderly, debilitated and often have advanced liver disease. Except for children it is done with local anesthesia, using one per cent novocain, without adrenalin, in sufficient amount to infiltrate all layers, down to and including the peritoneum. A stab wound, just the width of a number 10 Bard-Parker blade is made through the skin, deep enough to just nick the *rectus fascia*. The usual site elected is in the mid-line just below the umbilicus, except in children where we go through the rectus muscle. It is important, however, to select a site well away from any old operative

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scar. The pneumoperitoneum needle is then inserted into the peritoneal cavity, through this stab wound and swept around to test for adhesions. Air is then pumped into the abdominal cavity till it is quite tense, this lifts the wall away from the viscera and makes it safe to insert the trocar and sheath, which is popped through the tense wall just like going through a drum head. Any fluid present is then removed through the aspirator under suction, the visual telescope is installed and the abdomen explored. We have found it best to make a routine sweep of the abdomen around the clock, inspecting viscera and parietal peritoneum alike, then to go back and spend time inspecting organs suspected or pathology found. There are several maneuvers that facilitate visualization in certain instances. The table can be tilted from side to side, throwing the liver, spleen or tumor masses into view; or into the Trendelenburg position to drop the intestines out of the pelvis and bring the pelvic organs into view. The pelvic organs may be pushed up and orientation aided by an assistant manipulating the cervix through the vagina. In known cases of gastric carcinoma it is helpful to insert a modified Levine tube, with a light on the tip, into the stomach prior to the procedure. The stomach can then be inflated while being observed then the light turned on, on the tube and off on the visual telescope. This makes the stomach look just like a jack-o-lantern and will often give a very helpful idea as to the extent of infiltration and size of the lesion. Likewise inflating the colon with air may be most helpful. After exploration of the abdomen, biopsies are taken. These are always done under direct vision and the biopsy site coagulated when bleeding occurs, without injury to the tissue taken. The air is allowed to escape through the instrument, which is then withdrawn and the wound closed with a single skin suture.

After care is simple, the patient is requested to stay flat for six or eight hours until any air which did not escape is absorbed, to prevent shoulder pain. Fluids may be taken as soon as desired as well as food. Unless there has been abdominal pain prior to peritoneoscopy, there is usually little after pain, which can always be controlled with codein. The patient may go home the morning after peritoneoscopy is done.

Contraindications to peritoneoscopy are few. Obviously acute inflammatory conditions are definitely not to be considered. Age is not a contraindication, cases in our series range from six weeks to 88 years. Obstruction with marked abdominal distention is a contraindication as visualization would be poor and the help gained meager. Many of the cases that we are asked to do are emaciated, debilitated and would be considered extremely poor surgical risks.

The practical use of peritoneoscopy in the field of gastroenterology can be best illustrated by the following cases I have selected from my files done over the past 20 years. The majority of these were referred and had been worked up by good men. In abstracting these cases down to bare essentials because of time limitations, with the answer following, as demonstrated by peritoneoscopy and with the benefit of hindsight, the problems may seem overly simple at times.

They did not seem so to those referring them and were all done to answer some specific question. I do not intend to burden you with a lot of graphs and statistics in this paper and shall now pass on to the protocols.

Case 1:—A 40-year old, married female, who had vague gastrointestinal symptoms of over a year's duration. She had been studied at a large midwest clinic with negative findings except for achlorhydria, and was told she had "a nervous stomach"; however, sedation and HCl gave no relief. She developed progressive anorexia, nausea, vomiting, loss of weight and swelling of the abdomen. She entered a local hospital here and was studied thoroughly from December 28th through January 22nd without any conclusive findings. A gastroscopy done then showed several flattened papules, which were not diagnostic. At the time of peritoneoscopy on January 23rd, the only significant physical findings were emaciation and ascites. After removal of 5,700 c.c. of straw colored fluid, when the abdominal cavity was visualized, the parietal peritoneum was free from metastases, the liver was normal except for two small white implants, the stomach was markedly distended, the wall appeared to be thickened and on the greater curvature about one-third of the way down from the cardiac end two large, firm nodules were noted. These felt almost cartilaginous and on biopsy cut like malignant tissue. On visualization of the pelvis, the uterus was normal, no implants noted, the right ovary was normal in size and felt extremely hard, the left, was the same consistency, but larger than normal with several irregular nodules that appeared to be neoplastic, a biopsy was taken from this area. The peritoneoscopic diagnosis was neoplastic disease involving both ovaries and stomach, probably a Krukenberg tumor. The biopsy report corroborated this diagnosis.

Case 2:—A 66-year old, nervous, Italian widow, who had cramp-like pains in the upper abdomen for about one year. She had been afraid to eat and lost much weight. Though she had been constipated for years there had been no obstructive symptoms. She had had upper abdominal surgery many years before, the nature of which could not be learned. All laboratory work was within normal limits, x-ray studies of the gastrointestinal tract showed a nonspecific, deforming lesion involving the pylorus and a large colon. Her surgeon felt she was a very poor surgical risk and that she had a gastric malignancy with probable metastases to the liver. Physical examination at the time of peritoneoscopy was negative except for extreme emaciation. She appeared to be much older than the stated age, no masses or organs were palpable in the abdomen. On peritoneoscopy no evidence of malignancy was noted. There were extensive adhesions from the left lobe of the liver to the anterior chest wall. Just below the falciform ligament, the pyloric end of the stomach was seen, at this point the large bowel was puckered up by adhesions which started from the old postoperative scar and formed a dense band about one and a half inches in width, the whole mass of bowel and adhesions tied into the stomach. The colon was markedly dilated and enlarged. After several days preparation, the patient was operated and

the findings corroborated. She made an uneventful recovery and has been well since.

In any case of gastric malignancy, the problem of operability comes up. The rule of most surgeons is to "Open them up and see, you can always sew up any sized incision; that is the only safe way." True if there were no ancillary methods to help in this decision. Certainly a must if the patient has clinical and x-ray evidence of gastric retention, to give the patient relief. The main criteria as to operability, namely: "Are there metastases in the liver?"; "Extension to adjacent tissues?" and "How much stomach is involved?", can be answered in a large majority of cases by peritoneoscopy. Too many cases are opened surgically and closed because of liver metastases and extension to adjacent tissues, when the matter could have been determined in a simpler, less costly way, with far less discomfort to the patient. While I readily grant that it would not be feasible to peritoneoscope every case, there are a large number that should be done because of the likelihood that metastases to the liver has occurred. In my case records I have many hundreds of such instances. On the other hand there are also numerous cases, such as Case 2, in which we have been able to show the surgeon that he has an operable lesion and the surgery should be attempted even though the patient was an extremely poor surgical risk. I remember one such case in our earlier days, in an elderly, emaciated, debilitated, male, who had x-ray evidence of a large tumor of the stomach, thought to be inoperable, which Dr. Ruddock proved to be a benign leiomyoma.

In the field of liver disease, it is my feeling that peritoneoscopy is the most valuable single diagnostic weapon we have. There is a facetious saying that "One peek is worth two finesses". This is certainly true in liver disease, for a good look plus an adequate biopsy is worth a score of the laboratory procedures we ordinarily do in obtaining a liver profile. I have seen livers in advanced stages of cirrhosis, or almost completely replaced with carcinoma, show very normal profiles. I am also a firm believer in obtaining liver biopsies under direct vision. I know that the school which believes in needle biopsy, claims to get deeper into liver tissue and so get away from bizarre changes under the capsule. I am, however, very sure that the experienced peritoneoscopist will obtain a greater number of good biopsies and come forward with a much higher percentage of accurate diagnoses in the over all picture of liver disease. The gross picture in relation to other organs in the abdominal cavity is of great importance in making this diagnosis. If one feels a very deep section is needed and the needle employed, then it should be done under direct vision. Liver cysts are aspirated under direct vision. We find ideal for this purpose one of the solid tipped needles with a hole on the side, used in separating plasma. Occasionally a granulomatous peritonitis will be manifested by vague gastrointestinal symptoms. One such case was clinically diagnosed as probable atrophic cirrhosis after a careful study. Peritoneoscopy was requested to corroborate this, but proved it to be a granulomatous peritonitis. The biopsy proved the etiology to be *coccidioides imitus*.

It was apparently primary in the abdomen, lung findings not showing up for some months and the first case in the literature diagnosed antemortem.

I could spend several hours covering the different liver lesions that have been diagnosed by peritoneoscopy; but we do not have time for that. The following outline will indicate most of them:

DISEASES OF THE LIVER EXCLUSIVE OF CIRRHOSIS OR HEPATITIS

I. Anomalies:
   A. Riedel's Lobe
   B. Situs Inversus
   C. Hepatoptosis
II. Vascular Disturbances
   A. Hepatic Vein Thrombosis
   B. Portal Vein Thrombosis
      1. Suppurative
      2. Nonsuppurative
   C. Cruveillhier-Baumgartner's Syndrome
III. Hemachromatosis
IV. Hepatolienal Fibrosis
   Banti's Disease
V. Hepatolenticular Degeneration
   Wilson's Disease
VI. Benign Tumors
   A. Adenoma
      1. Single
      2. Multiple
   B. Cysts
      1. Solitary
      2. Multiple
VII. Neoplasms
   A. Primary Carcinoma
      1. Nodular
      2. Massive
      3. Diffuse
   B. Primary Sarcoma exceedingly rare.
   C. Secondary Carcinoma Metastatic
VIII. Specific Infections
   A. Syphilis
      1. Congenital
      2. Acquired Hepar Lobatum
   B. Tuberculosis
IX. Liver Abscesses
   A. Multiple
   B. Single
X. Parasitic Disease
   A. Echinococcus Cysts
   B. Amebic Abscess
   C. Other Organisms
XI. Metabolic
   A. Glycogen Storage Van Gierke's
   B. Fatty Liver
   C. Lipoidoses
      1. Gaucher's Disease
      2. Nieman Pick's Disease
      3. Xanthomatous Cirrhosis
   D. Amyloidosis

Case 3:—A 54-year old, male physician, who developed an ill-defined illness in 1943. A lymph gland biopsy showed Hodgkin's disease and there was good response to x-ray therapy. There were numerous recurrences always with good response to x-ray. In June 1952, he suddenly became ill, developed ascites and a high fever. There was no response to x-ray or antibiotics and in spite of all supportive therapy, he went down hill rapidly. It was felt he had a spread to the liver or a tuberculous peritonitis. At the time of peritoneoscopy he was lethargic to the point of stupor, emaciated, had marked ascites. The liver was not palpable. Peritoneoscopy showed a normal sized liver with the typical gross picture of the subacute stage of a portal cirrhosis. There was no gross evidence of lymphomatous infiltration of the liver or of tuberculosis. A biopsy was taken which was reported as portal cirrhosis. The patient was put on ACTH and later cortisone. He made a miraculous recovery; has gained a lot of weight; the ascites left and he is now actively engaged in practice again and well at this time.

Case 4:—A 47-year old priest, a diabetic of long standing, with arteriosclerotic heart disease, who had had several episodes of decompensation. His diabetes had been very hard to control, in spite of long periods of hospitalization. He was hospitalized finally, quite ill, mentally confused, liver enlarged and tender, function tests were poor. Peritoneoscopy was requested to determine if he had hemachromatosis. On visualization the liver was enlarged, coarsely granular, a peculiar brownish yellow color. On biopsy the liver substance was very firm, definitely rust colored, typical of hemachromatosis. Pathological diagnosis confirmed this. Patient died three months later, all organs contained iron deposits.

Case 5:—A 38-year old male, while at work had injured his left foot and developed a subsequent infection. Following this there was a complicating thrombophlebitis and then some migratory thrombi elsewhere in the body. About six months later he had a sudden rise in temperature, swelling of both legs and an unexplained ascites. He was seen by a top flight surgeon, who found a splenomegaly and thought that he had a portal thrombosis. I was asked to peritoneoscope him one and a half years after the original injury as the insurance company had postulated a cirrhosis of the liver, developing independent of his injury and wanted to disclaim care on that basis. A referee had recommended peritoneoscopy to prove the point. This was done and a normal gross appearing liver, slightly smaller than usual, found. The spleen was very large, dusky and showed areas of perisplenitis. A biopsy of the liver showed normal liver tissue and the insurance company with this evidence assumed responsibility for his care.

The picture of metastatic carcinoma of the liver is a frequent one in my field. This is often true in cases with vague abdominal symptoms, having no clear cut pattern or findings. It is often unsuspected. In many instances the biopsy we take may indicate the primary lesion and in many others the pathologist reports "Carcinoma highly undifferentiated". The problem, however,

is still answered in either instance and enables the attending physician to give relatives something definite; to arrange placement care and everyone to make some plans for the future even though they be dismal ones.

Case 6:—A 57-year old male, a heavy alcoholic throughout adult life, had been under treatment for cirrhosis of the liver. He had improved until two months prior to peritoneoscopy, when he developed ascites and his liver enlarged. It felt nodular and hard. The attending physician felt that he had developed a primary hepatoma or had a metastasis and requested peritoneoscopy to determine this. On visualization he had a typical far advanced atrophic cirrhosis. There was a very large metastatic carcinomatous nodule occupying most of the right lobe. The gallbladder was large and distended. Biopsy was reported as metastatic adenocarcinoma of liver, possibly arising in the gastrointestinal tract.

Case 7:—A six week-old baby girl, who was noted to have a large abdomen, two weeks after birth and on examination, was found to have a liver that was enlarging rapidly. She ate fairly well but it was thought that any weight gain was due to liver. At five weeks she was hospitalized and extensive laboratory studies made which did not aid in a diagnosis. At peritoneoscopy the liver filled practically the whole abdomen. It was pale in color, appeared lobulated and almost cystic but when biopsied the cystic lobules were filled with solid tumor-like tissue, and was reported as a malignant neuroblastoma, metastatic to the liver. Although the roentgenologist was reluctant to give x-ray therapy, he succumbed to pressure. There was a remarkable regression of the tumor and she did very well up to nine months, when she became acutely ill with fever, nausea and vomiting and died shortly after entering the hospital. Postmortem showed a diffuse hepatic fibrosis no evidence of the original pathology; broncho-pneumonia and diffuse myocarditis.

Case 8:—A 68-year old male, who had an eye removed two years prior because of melanotic sarcoma. Six weeks prior to peritoneoscopy he had an acute abdominal episode with pain resembling a perforating ulcer. After this quieted down it was noted his liver was enlarged and was increasing in size. There was no weight loss. On visualization the liver was tremendously enlarged, firm and coal black. The whole organ seemed to be replaced with this black tissue, no normal liver tissue being noted. Biopsy reported as malignant melanoma, metastatic to liver.

We cannot study the pancreas by peritoneoscopy. At times some help is obtained by inference as in the case of hydrops of the gallbladder. Occasionally, when there is a very large tumor, it may be diagnosed.

Case 9:—A 67-year old male, who had been under the care of a very thorough general man for 12 years. He had a very complete physical examination, blood count and urinalysis each year, the last one being March 1952. He had been well until the first week of February 1953, when he developed an infection in the right foot, a cellulitis, and a phlebitis. This was treated with anti-

biotics and subsided in a short time. No thrombosis was present. He continued to spike a fever however and was examined more carefully and a large mass was found in the upper abdomen that felt like a firm, nodular liver. There was no weight loss. All investigation was negative except for an elevated white count. It was felt he had a metastatic carcinoma in the liver and a peritoneoscopy was done on February 25th. On visualization the peritoneal surfaces were all studded with typical carcinomatous implants. The liver was found to be normal except for several metastatic nodules in the left lobe. On the right side, rising from deep in the abdomen just under the liver was a large irregular mass, which definitely appeared to be neoplastic. The peritoneoscopic diagnosis was retroperitoneal malignant tumor with metastases. Biopsies were taken from the parietal peritoneum and the main mass. They were reported as adenocarcinoma, pancreatic in origin.

The gallbladder can be visualized in the majority of cases done and we have seen much interesting pathology. The organ can not only be seen, but can be palpated with the tip of the examining telescope and can be transilluminated. Direct cholangiography under peritoneoscopic visualization has been done in a fairly large series of cases by a number of men, Royer being the principal advocate of this method. It has yielded much helpful information in cases of obscure obstructive jaundice. The incidence of complication due to bile leakage afterward has been small.

Case 10:—A 78-year old female, who had long standing arteriosclerotic heart disease with hypertension. She had, however, been quite well for the past two or three years. She had had intolerance to rich or fatty foods and had learned to stay away from them. No gallbladder studies had ever been done. On April 6, 1953 she had visited with friends in the country and ate a meal of very rich food. In four or five hours she developed nausea, vomiting and some upper abdominal pain, no fever. She was too ill to return to town until April 11th and since her physician was away would see no one until he came back on April 14th, by which time she felt all right. On examination, however, a very hard rounded mass was palpable. It was thought to be liver, but the abdominal wall was very thick and it was difficult to be sure. At the time of peritoneoscopy, which was done on April 18th there was slight tenderness present over the mass. On visualization the liver was found to be moderately enlarged and presented the gross picture of a very mild degree of hepatitis. There was no evidence of malignancy. The gallbladder was tremendously enlarged, elongated, the tip extending down to the iliac crest, with the patient in a moderate Trendelenburg position. The gallbladder wall was thickened, edematous and the whole organ tense, presenting a picture of subsiding inflammation. The patient did not desire surgery. A surgical consultant felt that in view of her age, hypertension and the fact that the process was definitely subsiding, she had made a wise decision. She was last seen January 1953 for routine check having no discomfort. The gallbladder was still palpable, but only about half as big as when first felt and

nontender. There had been no weight loss or gastrointestinal symptoms. She had been adhering rigidly to a low fat, bland diet.

The spleen is visualized when enlarged. The normal sized spleen is not usually seen. Diagnostic help in splenic disease is mainly in gross appearance, with relationship to the presence or absence of pathology that may exist in other abdominal organs. We rarely biopsy the spleen, the information obtained is usually of little help, the risk from hemorrhage greater than from biopsies on any other organ. The one exception is perhaps suspected amyloid disease.

Occasionally peritoneoscopy is of aid in disease of the colon. The presence of metastases in the liver can of course be determined. With colon disease, however, one is usually dealing with an obstructive lesion and surgery is necessary for that reason even though there may be metastases. The appendix is visualized in only about 15 per cent of cases. We feel that any case of suspected acute inflammatory disease is a contraindication for peritoneoscopy. I have, however, made the diagnosis of subsiding appendicitis. Particularly I am thinking of one case of a known pregnancy with abdominal pain and spotting, suspected of being an extrauterine pregnancy. Peritoneoscopy demonstrated to the obstetrician that he was dealing with an intrauterine pregnancy with a subsiding acute appendicitis. Conservative treatment was continued and she went to term.

Case 11:—An emaciated 52-year old male veteran, had been operated in the East for carcinoma of the rectum. A double barrelled colostomy had been done as a first stage. For some reason he came West and landed at Birmingham General Hospital. A large mass was palpable in the rectum. The patient gained weight in Birmingham General, while waiting for transfer of records. The records did not materialize and since considerable time had elapsed since the original surgery and there was no obstructive problem the surgeon wanted to know whether there were metastases in the liver or peritoneal cavity. Peritoneoscopy revealed a normal liver with no evidence of any metastatic carcinoma anywhere in the abdominal cavity. Subsequent surgery sustained this finding. Postoperative course was uneventful.

Case 12:—A 32-year old female, who had had a complete removal of all pelvic organs, with subsequent x-ray therapy in 1934 for papillary cyst-adenocarcinoma. In October 1936 she began having obstipation and then ribbon stools. Barium enema showed a constricting lesion of the sigmoid. Her surgeon felt she probably had a recurrence and she was reluctant to have surgery if such was the case. Peritoneoscopy showed a completely normal liver. All peritoneal surfaces were clean, there was no evidence, even in the pelvis of any recurrence. In the left side, at the termination of the round ligament, a dense band of adhesions which originated under the old postoperative scar appeared to wrap around and constrict the sigmoid. A sigmoidoscope was passed during the peritoneoscopy and the colon inflated with air. The band of adhesions definite-

ly acted as the obstructing mechanism. From the gross appearance it was felt there was not an annular carcinoma present. The patient was operated November 27, 1936 and peritoneoscopic findings corroborated. She had a normal evacuation the 4th postoperative day. Sigmoidoscopy done January 3rd, 1937 was negative. She has remained well to this day, with no evidence of recurrence of carcinoma or obstruction.

Conclusion

Peritoneoscopy is a most useful diagnostic adjunct in the field of gastroenterology. It is the most valuable single diagnostic procedure that we have for the study of liver disease, and when done by an experienced individual, with biopsy being obtained, will yield a higher percentage of correct diagnoses than all other diagnostic procedures put together. A few cases have been presented from those done in the past 20 years to illustrate these points.

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THE USE OF IRRADIATED PARAFFINS IN DIVERTICULOSIS*

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The common condition of multiple diverticula of the colon represents a definite disease of the colon wall. Invariably, whether an inflammatory condition (diverticulitis) exists in the colon wall or not, there is a varied array of microscopic pathology in the cellular structures, blood vessels and nervous tissues of the bowel wall. These may briefly be described as increase of laminated fibrous tissue, round cell infiltration, mask cells, cells with poor straining cytoplasm, vacuolated lymphocytoid cells, detached cells and blocked capillaries. The engraftation of an inflammatory process (diverticulitis) intensifies the scene and multiplies the cellular and tissue destruction, but the study of the colon tissue from 37 cases of diverticulosis and diverticulitis proves conclusively that we deal with a definite chronic disease throughout the area where the diverticula exist. It is most probable that the disease of the bowel existed before the diverticulum took place, or that it ensues simultaneously at the time the sacs are formed. After sufficient study, it seems reasonable to designate all the cases of colon diverticula as "diverticula disease" of the colon and that this is a disease in chronic character. This is the fact both whether in the quiescent or inflammatory state.

Studies of families suggests there is no hereditary factor connected with the condition. Mostly it occurs in middle-aged and older persons although it may be met with in young adults. There is suggestion that when the disease is established, in about half of the cases, the sacs increase in numbers and spread throughout the colon although in the majority of the cases they are in the left colon and especially in the sigmoid region. Often they increase in numbers with age, and careful studies of the stools and urine suggest that the cause of the acquired multiple diverticula is a long continued biotoxic condition of the colon contents. While the bacteriology of this is not always conclusive, the high incidence of the lytic forms of microorganisms strongly suggests that we deal with a chronic infection in the majority of cases. While the reaction to infection is deposition of white fibrous tissue, in these instances the fibrils are very small and sparse and too scattered to be of supportive effect on the vessel holes.

There also occurs a thinning-out of the normal connective tissue, and especially is this noticeable on the mesentery line of attachment. It is possible that lack of support along this line permits extrusion of the lining of the colon to occur with the production of diverticula to which intracolon tension from gas would be a factor of sac formation.

This is not the place to go into detail as to the dietetic and biological handling of diverticulosis. Other than those measures that cause more normal bowel

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eliminations in constipated persons, there is no value to dietetic measures excepting in diverticulitis when, for the time being, logic suggests that fluid and bland diets would be more in order. The disease in the gut wall cannot be cured or such symptoms as may be present are not relieved by dietetic control. The same may be stated regarding biologic treatments (Vaccines, sera, phages, etc.) or medicinal handling (sulfa drugs, antibiotics). When the diverticula are established, the cytology in the gut wall is destroyed and for this there are no curative remedies. The most one may hope to attain is to modify the pathology in the colon wall, assure better bowel elimination, keep the sacs empty of long retained feces, and employ a remedy that has a beneficial selective action on the biotoxic state of the bowel contents.

If one irradiates petroleum jelly with a quartz lamp for 15 to 30 minutes, two things happen to the jelly. When it becomes solid again, (and it's best to radiate the jelly when heated to a fluid), the jelly changes in physical character. It becomes brittle, so to speak, in that its coherence is lost. This broken-up state (especially if a little spray oil had been added to the soft paraffin originally) mixes and incorporates itself with feces and does not float around like mineral oil does. The addition of spray oil assists the paraffin to go into a more fluid state at body temperature, a desirable factor to keep the sacs empty of long sustained feces. Irradiated fluid mineral oil is a poor substitute to the jelly and does not retain the radiation long enough to meet the second point, namely, a destructive effect on the low grade infection that is present in these cases. The destructive effect of irradiated vaseline on bacteria is superior for the purpose to any of the biotics or sulfa drugs, and is in no sense bacteriostatic but distinctly bacteriocidal. With this jelly we have three factors at work; first, the stimulation of better bowel elimination; second, the cleansing of the sacs; and third, a bacteriocidal effect on the low grade infection that is present.

To this product is added 20 per cent of beta lactose sugar to stimulate the resisting forms of gram negative bacteria, and 4 per cent of oleum ricini which, in small doses and taken steadily, has a beneficial effect on the colon mucosa, especially that of the sigmoid region.

Taken in tablespoonful doses on an empty stomach once or twice a day, the abdominal symptoms of diverticular disease of the colon soon disappear, and in those who take this remedy more or less steadily there is commonly an improvement in general health. The rule is that the diverticula not only do not increase but often decrease in size and numbers, and I have had several cases where it has not been possible to demonstrate any diverticula present after taking the irradiated vaseline for several months' time. In fastidious people the preliminary use of a chocolate candy, especially the non-sweetened type, helps in overcoming the feeling of the oleogenous mixture in the mouth. I would like to conclude by saying that we have here almost a specific for the handling of diverticula disease, perhaps the first logical treatment that has been advanced; and I state this after using the remedy for a number of years.

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VISIT THE EXHIBITS

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GERWIG, WALTER H., Jr., A.B., M.D., Washington, D. C. Assistant Clinical Professor of Surgery, George Washington University, School of Medicine; Chief of Proctology Clinic, George Washington University Hospital. *(p. 26)*

GIBB, WILLIAM T., A.B., M.D., Washington, D. C. Assistant Clinical Professor of Medicine, George Washington University, School of Medicine; Director of Medicine, Suburban Hospital, Bethesda, Md. *(p. 12)*

GLASS, GEORGE B. JERZY, M.D., New York, N. Y. Associate Professor of Medicine, New York Medical College; Associate Attending Physician, Flower and Fifth Avenue Hospitals; Associate Visiting Physician, Metropolitan and Bird S. Coler Hospitals. *(p. 17)*

GLENN, JOSEPH BURTON, M.D., F.A.C.P., Washington, D. C. *(p. 10)*

GOLDBLOOM, A. ALLEN, M.D., F.A.C.P., F.A.C.G., New York, N. Y. Associate Professor of Medicine, New York Medical College, Flower and Fifth Avenue Hospitals; Attending Physician, Metropolitan, Bird S. Coler and Beth Israel Hospitals; Associate Attending Physician, Flower Fifth Avenue Hospital. *(p. 19)*

GORDON, JULES D., M.D., F.A.C.S., F.A.C.C., F.A.C.G., New York, N. Y. Instructor in Surgery, New York Medical College; Attending Surgeon, Beth David Hospital; Associate Surgeon, Beth Israel Hospital. *(p. 17)*

GRAY, SEYMOUR J., M.D., Ph.D., F.A.C.P., Boston, Mass. Assistant Professor of Medicine, Harvard Medical School; Senior Associate in Medicine, Peter Bent Brigham Hospital. *(p. 21)*

HADRA, EDMUND, M.D., New York, N. Y. *(p. 19)*

HAVENS, W. PAUL, Jr., A.B., M.D., Philadelphia, Pa. Associate Professor of Medicine, Jefferson Medical College of Philadelphia; Chief, Section of Infectious Diseases, Pennsylvania Hospital. *(p. 22)*

HAYNES, JOHN D., M.D., Baltimore, Md. Resident in Surgery, Baltimore City Hospital. *(p. 22)*

HEATON, LEONARD A., Maj. Gen., U.S.A., Washington, D. C., Commanding General, Walter Reed Army Hospital. *(pp. 12, 23)*

HERSHENSON, MORRIS A., B.S., M.D., F.A.C.G., Pittsburgh, Pa. Associate Professor of Medicine, University of Pittsburgh, School of Medicine *(p. 18)*

HOWARD, JOHN T., B.S., M.D., Baltimore, Md. Assistant Professor of Medicine, The Johns Hopkins University, School of Medicine. *(p. 15)*

HUFNAGEL, CHARLES A., B.S., M.D., F.A.C.S., Washington, D. C. Associate Professor of Surgery and Professor of Surgical Research, Georgetown University Medical Center. *(p. 12)*

HUGHES, CARL W., Lt. Col., M.C., Washington, D.C. Chief, Vascular Surgery, Walter Reed Army Hospital; Deputy Director, Division of Surgery, Army Medical Service Graduate School. *(p.23)*

HUTTON, SAMERILL, M.D., New Orleans, La. *(p. 14)*

IMBODEN, C. A., Jr., B.S., M.D., Bethesda, Md. Laboratory of Clinical Investigation, National Microbiological Institute, Clinical Center, National Institutes of Health. *(p. 11)*

JAHNKE, EDWARD J., Jr., Maj., M.C., Washington, D. C. Resident in Thoracic Surgery, Walter Reed Army Hospital. *(pp. 12, 23)*

JAMES, PHILIP R., B.A., M.D., Bethesda, Md. Resident in Medicine, U. S. Naval Hospital, Bethesda, Md. *(p. 9)*

JAY, JACK B., Maj., M.C., Washington, D. C. Chief Resident, General Surgical Service, Walter Reed Army Hospital. *(p. 24)*

JOHNSON, FRANK B., M.D., Washington, D. C. Armed Forces Institute of Pathology. *(p. 9)*

JOHNSON, GAIL, M.D., New Orleans, La. *(p. 14)*

KAPLAN, I. W., M.D., New Orleans, La. Assistant Clinical Professor of Surgery, Louisiana State University, School of Medicine; Chief Surgeon, Touro Infirmary; Senior Visiting Surgeon, Charity Hospital of Louisiana at New Orleans. *(p. 14)*

KARLSON, KARL E., M.D., Ph.D., Brooklyn, N. Y. Associate Professor of Surgery, State University of New York, College of Medicine. *(p. 25)*

VISIT THE EXHIBITS

KEITH, LUTHER M., Jr., M.D., Columbus, Ohio. Assistant Professor of Surgery, University of Ohio, School of Medicine. *(p. 25)*

KRANTZ, JOHN C., Jr., Ph.D., Baltimore, Md. Professor of Pharmacology, University of Maryland, School of Medicine. *(p. 17)*

KRAUSE, LOUIS, M.D., Baltimore, Md. Professor of Clinical Medicine, University of Maryland, School of Medicine; Chief of Medicine, Lutheran and City Hospitals. *(p. 15)*

LODMELL, ELMER A., Col., M.C., Washington, D. C. Chief, Radiological Service, Walter Reed Army Hospital; Chief Radiological Consultant, Office of the Surgeon General, Department of the Army. *(p. 23)*

LOPEZ, O., M.D., Havana, Cuba. University Hospital. *(p. 19)*

LYNCH, ROBERT C., M.D., New Orleans, La. Assistant Professor of Clinical Surgery, Tulane University of Louisiana, School of Medicine; Staff Member, Ochsner Foundation Hospital. *(p. 14)*

MANSBERGER, ARLIE R., Jr., M.D., Baltimore, Md. Resident in General Surgery, University Hospital. *(p. 16)*

MARTIN, LAY, B.S., M.D., Baltimore, Md. Assistant Professor of Medicine, The Johns Hopkins University, School of Medicine. *(p. 16)*

MC CULLOUGH, NORMAN B., B.S., M.S., Ph.D., M.D., Bethesda, Md. Chief, Laboratory of Clinical Investigation, National Microbiological Institute; Chief Infectious and Parasitic Disease Service, National Institutes of Health. *(p. 10)*

MEDOFF, JOSEPH, B.S., M.D., Philadelphia, Pa. Assistant in Medicine, Jefferson Medical College of Philadelphia. *(p. 14)*

MILANES, FERNANDO, M.D., F.A.C.G., Havana, Cuba. Professor of Medicine, University of Havana, School of Medicine. *(p. 19)*

MITCHELL, ROBERT EDGAR, Jr., A.B., M.D., New Orleans, La. Instructor in Medicine, Tulane University of Louisiana. *(p. 13)*

MONAT, HENRY A., A.B., M.D., F.A.C.P., F.A.C.G., Washington, D. C. Formerly Associate Clinical Professor of Medicine, Georgetown University, School of Medicine. *(p. 10)*

MORA, LIDIO, M.D., Havana, Cuba. Department of Gastroenterology, Mercedes Hospital. *(p. 19)*

MORGAN, ZACH R., M.D., F.A.C.G., Baltimore, Md. Assistant Professor of Gastroenterology, University of Maryland, School of Medicine; Chief, Department of Medicine, Church Home and Hospital; Physician to the University Hospital; Women's, Union Memorial and Maryland General Hospitals. *(p. 16)*

MORRISON, SAMUEL, A.B., M.D., Baltimore, Md. Associate Professor of Medicine and Associate Professor of Gastroenterology, University of Maryland, School of Medicine. *(p. 25)*

NIEBURGS, H. E., M.D., Brooklyn, N. Y. Director, Cytology Center, Beth-El Hospital. *(p. 18)*

OSBOURN, RAYMOND A., M.D., Washington, D. C. Assistant Professor of Dermatology, Georgetown University, School of Medicine. *(p. 24)*

PACK, GEORGE T., B.S., M.D., LL.D., New York, N. Y. Clinical Professor of Surgery, New York Medical College; Associate Professor of Clinical Surgery, Cornell University, School of Medicine; Attending Surgeon, Memorial Hospital; Pack Medical Group. *(p. 13)*

PALMER, EDDY D., Lt. Col., M.C., Washington, D. C. Chief, Gastroenterology Service, Walter Reed Army Hospital; Adjunct Instructor in Medicine, Georgetown University, School of Medicine. *(pp. 12, 23, 24)*

PAULSON, MOSES, B.S., M.D., F.A.C.P., Baltimore, Md. Assistant Professor of Medicine, The Johns Hopkins University, School of Medicine; Physician, The Johns Hopkins Hospital; Consultant in Digestive Diseases, Diagnostic Clinic and Private Out-Patient Services, The Johns Hopkins Hospital. *(p. 22)*

PERKEL, LOUIS L., B.S., M.D., F.A.C.P., F.A.C.G., Jersey City, N. J. Director, Department of Gastroenterology, Jersey City Medical Center; Consultant Gestroenterologist, Margaret Hague Maternity, North Hudson, St. Francis and Pollak Chest Disease Hospitals. *(p. 18)*

PLOUGH, IRVIN C., Maj., M.C., Washington, D. C. Chief, Department of Hepatic and Metabolic Diseases, Army Medical Service Graduate School; Medical Service, Walter Reed Army Hospital. *(p. 23)*

VISIT THE EXHIBITS

POMERANZE, JULIUS, M.D., New York, N. Y. *(p. 19)*

PRUITT, FRANCIS W., Col., M.C., Washington, D. C. Chief, Department of Medicine, Walter Reed Army Hospital. *(p. 24)*

PUESTOW, KARVER L., M.D., F.A.C.G., Madison, Wisc. Professor of Medicine, University of Wisconsin, School of Medicine; Attending Physician, University Hospitals. *(p. 21)*

PUTNEY, F. J., B.S., M.D., Philadelphia, Pa. Associate Professor of Laryngology and Bronchoesophagology, Jefferson Medical College of Philadelphia. *(p. 21)*

RECHTSCHAFFEN, JOSEPH S., M.D., New York, N. Y. *(p. 19)*

REES, CHARLES W., B.S., M.A., Ph.D., Bethesda, Md. Chief, Section on Protozoal Diseases, Microbiological Institute, Laboratory of Tropical Diseases. *(p. 11)*

RICH, MARILYN, B.S., New York, N. Y. *(p. 17)*

RIPSTEIN, CHARLES B., M.D., C.M., F.A.C.S., F.R.C.S., Brooklyn, N. Y. Professor of Surgery, State University of New York, College of Medicine. *(p. 19)*

SACKS, MILTON S., B.S., M.D., Baltimore, Md. Associate Professor of Medicine and Head of the Department of Clinical Pathology, University of Maryland, School of Medicine. *(p. 17)*

SCOTT, NORMAN M., Jr., Maj., M.C., Washington, D. C. Resident in Gastroenterology, Walter Reed Army Hospital. *(p. 23)*

SEXTON, ROY LYMAN, M.D., Washington, D. C. *(p. 10)*

SHAIKEN, JOSEPH, M.D., M.Sc., F.A.C.G., Milwaukee, Wisc. Associate Professor of Medicine, Marquette University Medical School; Chief, Department of Medicine, Milwaukee County Hospital; Director of Medical Education, Mt. Sinai Hospital. *(p. 13)*

SHALLOW, THOMAS A., M.D., F.A.C.S., LL.D., Philadelphia, Pa. Professor of Surgery and Head of Department; Jefferson Medical College of Philadelphia; Attending Surgeon, Jefferson Medical College and Hospital. *(p. 13)*

SHUTKIN, MICHAEL W., B.A., M.D., F.A.C.G., Milwaukee, Wisc. Assistant Clinical Professor of Medicine, Marquette University, School of Medicine; Senior Attending Gastroenterologist and Gastroscopist, Milwaukee County, Mt. Sinai, and St. Luke's Hospitals. *(p. 18)*

SMITH, ROBERT S., M.D., Washington, D. C. Attending Staff, Emergency Hospital and Bethesda Suburban Hospitals. *(p. 14)*

SNAPPER, I., M.D., Ph.D., Brooklyn, N. Y. Director of Medical Education, Beth-El Hospital. *(p. 21)*

SPAIN, DAVID M., M.D., Brooklyn, N. Y. Assistant Professor of Pathology, Columbia University, College of Physicians and Surgeons; Director of Laboratories, Beth-El Hospital. *(p. 26)*

SPICKNALL, CHARLES G., M.D., Baltimore, Md. Chief, Medical Service, United States Public Health Service Hospital. *(p. 11)*

TALBOTT, EDMUND J., M.D., D.T.M. & H., Bethesda, Md. Laboratory of Tropical Diseases, National Microbiological Institute, Clinical Center, National Institutes of Health. *(p. 11)*

TERRY, LUTHER L., B.S., M.D., Bethesda, Md. Assistant Professor of Medicine, The Johns Hopkins University, School of Medicine; Chief, Clinic of General Medicine and Experimental Therapeutics, National Heart Institute. *(p. 11)*

THOMAS, J. EARL, M.D., Ph.D., Philadelphia, Pa. Professor of Physiology, Jefferson Medical College of Philadelphia. *(p. 9)*

TOBIE, JOHN E., Ph.D., Bethesda, Md. Laboratory of Tropical Diseases, National Institutes of Health. *(p. 11)*

TRIMBLE, I. RIDGEWAY, A.B., M.D., Baltimore, Md. Associate Professor of Surgery, The Johns Hopkins University, School of Medicine; Professor of Clinical Surgery, University of Maryland, School of Medicine. *(p. 16)*

TROAST, LEONARD, B.S., M.D., Jersey City, N. J. Attending Physician, Jersey City Medical Center. *(p. 18)*

WAGNER, FREDERICK B., Jr., A.B., M.D., F.A.C.S., Philadelphia, Pa. Associate Professor of Surgery and Assistant Surgeon, Jefferson Medical College and Hospital. *(p. 13)*

WANGENSTEEN, OWEN H., B.A., M.D., Ph.D., Minneapolis, Minn. Professor and Chairman, Department of Surgery, University of Minnesota, School of Medicine. *(pp. 21, 24)*

VISIT THE EXHIBITS

WEINSTEIN, VERNON A., A.B., M.D., New York, N. Y. Assistant Visiting Surgeon, The Mt. Sinai Hospital. *(p. 22)*

WELCH, GEORGE E., B.S., M.D., New Orleans, La. Instructor in Medicine, Tulane University of Louisiana, School of Medicine; Junior, Department of Gastroenterology, Touro Infirmary; Visiting Physician, Charity Hospital of Louisiana at New Orleans; Visiting Staff, Sara Mayo Hospital. *(p. 13)*

WERNER, ABRAHAM, M.D., F.A.C.G., New York, N. Y. Chief, Internal Medicine Section, Brooklyn Regional Office, Veterans Administration. *(p. 18)*

WILLNER, VICTOR, M.D., F.A.C.G., Hempstead, N. Y. Assistant Attending, Meadowbrook Hospital. *(p. 18)*

WRIGHT, WILLARD H., M.S., D.V.M., Ph.D., Bethesda, Md. Chief, Laboratory of Tropical Diseases and Assistant Director, National Microbiological Institute, National Institute of Health; Lecturer in Medical Zoology, George Washington University, School of Medicine; Visiting Lecturer in Tropical Public Health, Harvard University. *(p. 10)*

Business Sessions

SUNDAY AFTERNOON, 24 OCTOBER 1954

4:00 P.M.

Meeting of the Board of Trustees of the American College of Gastroenterology—Louis Seize Room.

6:00 P.M.

Board of Trustees Banquet—Blue Room.

MONDAY AFTERNOON, 25 OCTOBER 1954

5:00 P.M.

Annual Meeting of the American College of Gastroenterology—Terrace Room.

6:00 P.M.

Annual Meeting of the National Gastroenterological Association—Terrace Room.

TUESDAY AFTERNOON, 26 OCTOBER 1954

5:00 P.M.

Organizational Meeting of the Board of Governors.

WEDNESDAY AFTERNOON, 27 OCTOBER 1954

12:30 P.M.

Luncheon Meeting of the Board of Trustees—Louis Seize Room.

VISIT THE EXHIBITS

SCIENTIFIC SESSIONS

FIRST SESSION

MONDAY MORNING, 25 OCTOBER 1954

SIGURD W. JOHNSEN, M.D., F.A.C.G., President, National Gastroenterological Association, presiding.

9:00 A.M.

1. "Chronic Idiopathic Jaundice with Unidentified Pigment in Liver Cells".

Speakers

DR. I. N. DUBIN, Washington, D. C. (By invitation) and DR. FRANK B. JOHNSON, Washington, D. C. (By invitation).

9:20 A.M.

2. "The Gradient Theory Versus the Reflex Theory of Intestinal Peristalsis".

Speaker

DR. J. EARL THOMAS, Philadelphia, Pa. (By invitation).

9:40 A.M.

3. "Gastrointestinal Allergy—A Review and Case Report".

Speaker

DR. PHILIP R. JAMES, Bethesda, Md. (By invitation).

10:00 A.M.

4. "Mild Ulcerative Colitis".

Speaker

DR. JAMES F. BISHOP, Davenport, Iowa (By invitation).

10:20 A.M.

General discussion of papers 1 to 4.

10:30 A.M. Recess.

VISIT THE EXHIBITS

10:45 A.M.

5. "PANEL DISCUSSION ON TWENTY-FIVE YEARS' OBSERVATION OF THE GALLBLADDER CONTROVERSY".

Moderator: DR. ROY LYMAN SEXTON, Washington, D. C. (By invitation).

Participants:

a. "Our Most Serious Differential Diagnosis, The Coronary Heart and Gallbladder Disease".
DR. JOSEPH BURTON GLENN, Washington, D. C. (By invitation).

b. "Medical Aspects of Noncalculus Gallbladder Disease".
DR. J. EDWARD BERK, Detroit, Mich. (By invitation).

c. "Surgical Approach to the Problems of Gallbladder Disease".
DR. HARRY LEE CLAUD, Washington, D. C. (By invitation).

SECOND SESSION

MONDAY AFTERNOON, 25 OCTOBER 1954

Exhibits will be open from 1:30 P.M. until closing.

JAMES T. NIX, M.D., F.A.C.G., Vice-President, National Gastroenterological Association, presiding.

2:00 P.M.

6. "PANEL DISCUSSION ON AMEBIASIS".

a. "Some Problems in Amebiasis as Viewed by the Practitioner".
DR. HENRY A. MONAT, Washington, D. C., Organizer of the Panel.

b. Introduction of the Moderator.

Moderator: DR. NORMAN B. MCCULLOUGH, Bethesda, Md. (By invitation).

Participants:

c. "Clinical and Laboratory Approach to Problems in Amebiasis".

1. "The Research Approach".
DR. NORMAN B. MCCULLOUGH, Bethesda, Md. (My invitation).

2. "The Laboratory Approach".
DR. WILLARD H. WRIGHT, Bethesda, Md. (By invitation).

VISIT THE EXHIBITS

d. "Factors Related to the Pathogenicity of Endameba Histolytica".
DR. CHARLES W. REES, Bethesda, Md. (By invitation).

e. "Variations Between the Manifestations of Amebiasis in Temperate and Tropical Zones".
DR. CLARENCE A. IMBODEN, JR., Bethesda, Md. (By invitation).

3:05 P.M. Recess to visit the commercial, technical and scientific exhibits.

3:45 P.M.
PANEL DISCUSSION ON AMEBIASIS (Continued).
f. "Considerations Relative to the Diagnosis of Amebiasis".

1. "Clinical Considerations".
DR. LUTHER L. TERRY, Bethesda, Md. (By invitation).

2. "Parasitological Considerations".
DR. JOHN E. TOBIE, Bethesda, Md. (By invitation).

3. "Serological Considerations".
JOHN BOZICEVICH, Bethesda, Md. (By invitation).

g. "Treatment of Amebiasis".

1. "Present Day Concepts".
DR. EDMUND J. TALBOTT, Bethesda, Md. (By invitation).

2. "Experience in U.S.P.H.S. Hospital, Baltimore".
DR. LUTHER L. TERRY, Bethesda, Md. (By invitation) and DR. CHARLES G. SPICKNALL, Baltimore, Md. (By invitation).

4:30 P.M.
General Discussion.

5:00 P.M.
ANNUAL MEETING OF THE AMERICAN COLLEGE OF GASTROENTEROLOGY—Terrace Room.

6:00 P.M.
ANNUAL MEETING OF THE NATIONAL GASTROENTEROLOGICAL ASSOCIATION—Terrace Room.

6:30 P.M.
CONVOCATION: Presentation of Certificates—West Ball Room.
See special program.

VISIT THE EXHIBITS

8:00 P.M.

PRESIDENT'S ANNUAL RECEPTION—Louis Seize Room. Sponsored by Winthrop-Stearns, Inc. (admission by card only, to be obtained at the Convocation Ceremony).

THIRD SESSION

TUESDAY MORNING, 26 OCTOBER 1954

8:30 A.M. *Coffee and doughnuts will be served in the Exhibit Area with the compliments of the Association.*

C. WILMER WIRTS, M.D., F.A.C.G., Vice-President, National Gastroenterological Association, presiding.

9:00 A.M.

7. "SYMPOSIUM ON ESOPHAGEAL VARICES".

Moderator: DR. IRVING B. BRICK, Washington, D. C. (By invitation).

Participants:

a. "Mucosal and Organ Prolapse at the Esophagogastric Junction".
LT. COL. EDDY D. PALMER, M.C., Washington, D.C. (By invitation).

b. "Surgical Treatment of Portal Hypertension".
DR. CHARLES A. HUFNAGEL, Washington, D. C. (By invitation).

c. "Subject to be Announced".
MAJ. EDWARD J. JAHNKE, M.C., Washington, D. C. (By invitation).

10:20 A.M.
General discussion.

10:30 A.M. Recess to visit commercial, technical and scientific exhibits.

11:00 A.M.

8. "Gastric Decompensation".

Speaker
DR. WILLIAM T. GIBB, Washington, D. C. (By invitation).

VISIT THE EXHIBITS

11:20 A.M.

9. "Gastric Ulcer—Better Criteria for Benignancy and Malignancy".

Speakers

DR. DONOVAN C. BROWNE, New Orleans, La. (By invitation), DR. GEORGE E. WELCH, New Orleans, La. (By invitation) and DR. ROBERT EDGAR MITCHELL, JR., New Orleans, La. (By invitation).

11:40 A.M.

10. "Gastroenterological Aspects of Meckel's Diverticulum".

Speakers

DR. FREDERICK B. WAGNER, JR., Philadelphia, Pa. (By invitation), DR. THOMAS A. SHALLOW, Philadelphia, Pa. (By invitation) and DR. SHERMAN A. EGER, Philadelphia, Pa. (By invitation).

12:00 Noon

Discussion of paper 10 to be opened by DR. C. WILMER WIRTS, Philadelphia Pa.

12:30 P.M.

LUNCHEON—West Ball Room. Sponsored by Burton, Parsons & Co. (admission by card only, to be obtained at the registration desk).

Speaker

DR. GEORGE T. PACK, New York, N. Y. (By invitation).

FOURTH SESSION

TUESDAY AFTERNOON, 26 OCTOBER 1954

ARTHUR A. KIRCHNER, M.D., F.A.C.G., Vice-President, National Gastroenterological Association, presiding.

2:00 P.M.

11. "The Female Patient with Duodenal Ulcer".

Speaker

DR. JOSEPH SHAIKEN, Milwaukee, Wisc.

VISIT THE EXHIBITS

2:30 P.M.

12. "Unusual Types of Gastrointestinal Hemorrhage".

Speakers

DR. JOSEPH MEDOFF, Philadelphia, Pa. (By invitation), DR. O. F. DE GOUVEIA, Philadelphia, Pa. (By invitation) and DR. PAUL C. CIMVEH, Philadelphia, Pa. (By invitation).

2:40 P.M.

13. "Operative Cholangiography".

Speaker

DR. I. W. KAPLAN, New Orleans, La. (By invitation).

3:00 P.M.

General discussion of papers 11 to 13.

3:10 P.M. Recess to visit commercial, technical and scientific exhibits.

3:40 P.M.

14. "The Nonsurgical Mortality of Carcinoma of the Stomach".

Speaker

DR. FREDERICK FITZHERBERT BOYCE, New Orleans, La. (By invitation).

4:00 P.M.

15. "Carcinoma of the Cecum".

Speakers

DR. ROBERT C. LYNCH, New Orleans, La. (By invitation), DR. SAMERILL HUTTON, New Orleans, La. (By invitation) and DR. GAIL JOHNSON, New Orleans, La. (By invitation).

4:20 P.M.

16. "Cancer of the Distal Colon and Rectum—Treatment by Extended Resection".

Speakers

DR. GARNET W. AULT, Washington, D. C. (By invitation), DR. ROBERT S. SMITH, Washington, D. C. (By invitation) and DR. ALEJANDRO F. CASTRO, Washington, D. C. (By invitation).

VISIT THE EXHIBITS

5:00 P.M.
ORGANIZATIONAL MEETING OF THE BOARD OF GOVERNORS.

7:00 P.M.
ANNUAL BANQUET—West Ball Room, The Shoreham, Washington, D. C.

FIFTH SESSION

WEDNESDAY MORNING, 27 OCTOBER 1954

8:30 A.M. *Coffee and doughnuts will be served in the Exhibit Area with the compliments of the Association.*

FRANK J. BORRELLI, M.D., F.A.C.G., Vice-President, National Gastroenterological Association, presiding.

9:00 A.M.
17. "The Management of Instrumental Perforation of the Esophagus".

Speaker
DR. JOHN TILDEN HOWARD, Baltimore, Md. (By invitation).

9:20 A.M.
18. "The Internist and Gastroenterology".

Speaker
DR. LOUIS KRAUSE, Baltimore, Md. (By invitation).

9:40 A.M.
19. "Cirrhosis of the Liver—its Relationship to Cholesterosis of the Gallbladder, Gallstones and Other Associated Conditions".

Speakers
DR. MAURICE FELDMAN, Baltimore, Md. (By invitation) and DR. MAURICE FELDMAN, JR., Baltimore, Md. (By invitation).

10:00 A.M.
General discussion of papers 17 to 19.

10:10 A.M. Recess to visit commercial, technical and scientific exhibits.

VISIT THE EXHIBITS

10:40 A.M.

20. "Some Studies of the Electrolytes of the Gastric Mucosa and Muscularis".

Speaker

DR. LAY MARTIN, Baltimore, Md. (By invitation).

11:00 A.M.

21. "Treatment of Recurring Pancreatitis".

Speaker

DR. I. RIDGEWAY TRIMBLE, Baltimore, Md. (By invitation).

11:20 A.M.

22. "Headaches Associated with Gastrointestinal Disturbances".

Speaker

DR. ZACH R. MORGAN, Baltimore, Md.

11:40 A.M.

General discussion of papers 20 to 22.

12:00 Noon

LUNCHEON MEETING—Board of Trustees—Louis Seize Room.

SIXTH SESSION

WEDNESDAY AFTERNOON, 27 OCTOBER 1954

ROY UPHAM, M.D., F.A.C.G., Secretary-General, National Gastroenterological Association, presiding.

2:00 P.M.

23. "Some Practical Considerations of Massive Gastrointestinal Hemorrhage".

Speakers

DR. C. REID EDWARDS, Baltimore, Md. (By invitation) and DR. ARLIE R. MANSBERGER, JR., Baltimore, Md. (By invitation).

VISIT THE EXHIBITS

2:20 P.M.

24. "Blood Diseases and the Gastrointestinal Tract".

Speaker

Dr. Milton S. Sacks, Baltimore, Md. (By invitation).

2:40 P.M.

25. "Pharmacology of the Spasmolytic Drugs".

Speaker

Dr. John C. Krantz, Baltimore, Md. (By invitation).

3:00 P.M.

General discussion on papers 23 to 25.

3:10 P.M. Recess to visit commercial, technical and scientific exhibits.

3:40 P.M.

26. "Gastroscopic Observations of the Anticholinergic Effect of Pro-Banthine on Gastric Motility and Pyloric Function".

Speaker

Dr. Harry Barowsky, New York, N. Y.

4:00 P.M.

27. "The Early Operative Treatment for Gastric Hemorrhage".

Speaker

Dr. Jules D. Gordon, New York, N. Y.

4:20 P.M.

28. "Comprehensive Testing of Gastric Secretory Function".

Speakers

Dr. George B. Jerzy Glass, New York, N. Y. and Marilyn Rich, New York, N. Y. (By invitation).

4:40 P.M.

General discussion on papers 26 to 28.

VISIT THE EXHIBITS

SEVENTH SESSION

WEDNESDAY EVENING, 27 OCTOBER 1954

ANTHONY BASSLER, M.D., F.A.C.G., Honorary President, National Gastroenterological Association, presiding.

8:00 P.M.

29. "Reflux Esophagitis".

Speaker

DR. MORRIS A. HERSHENSON, Pittsburgh, Pa.

8:15 P.M.

30. "A New Cytological Method as an Aid in the Diagnosis of Gastric Malignancy".

Speakers

DR. VICTOR WILLNER, New York, N. Y., DR. H. E. NIEBURGS, Brooklyn, N. Y. (By invitation) and DR. B. FUCHS, Hempstead, N. Y. (By invitation).

8:30 P.M.

31. "Medical Management of Gastric Ulcer".

Speakers

DR. SIDNEY FIERST, Brooklyn, N. Y., DR. ABRAHAM WERNER, New York, N. Y. and DR. JAMES GABRIEL, Brooklyn, N. Y. (By invitation).

8:45 P.M.

32. "Ileal Prolapse Simulating Cecal Polyp".

Speakers

DR. LOUIS L. PERKEL, Jersey City, N. J. and DR. LEONARD TROAST, Jersey City, N. J.

9:00 P.M.

33. "The Clinical Value of Bilirubin Determinations in Routine Urinalysis with an Improved Method".

Speakers

DR. MICHAEL W. SHUTKIN, Milwaukee, Wisc. and DR. DONALD CAINE, Milwaukee, Wisc. (By invitation).

VISIT THE EXHIBITS

9:15 P.M.

34. "Bleeding Esophageal Varices".

Speaker

Dr. Charles B. Ripstein, Brooklyn, N. Y.

9:30 P.M.

35. "Newer Clinical and Laboratory Studies in the Aged. III. Urinary Test for Gastric Secretion of Hydrochloric Acid in Patients 80 to 100 Years of Age".

Speakers

Dr. A. Allen Goldbloom, New York, N. Y., Dr. Edmund G. Hadra, New York, N. Y. (By invitation), Dr. Julius Pomeranze, New York, N. Y. (By invitation) and Dr. Joseph S. Rechtschaffen, New York, N. Y.

9:45 P.M.

36. "Intravenous Cholecysto-Cholangiography with Biligrafin".

Speakers

Dr. Fernando Milanes, Havana, Cuba, Dr. Fidel Aguirre, Havana, Cuba (By invitation), Dr. Lidio Mora, Havana, Cuba (By invitation), Dr. F. Conde, Havana, Cuba (By invitation) and Dr. O. Lopez, Havana, Cuba (By invitation).

10:00 P.M.

37. "The Tubeless Gastric Analysis in the Study of Gastric Secretion in Partial Gastrectomy".

Speakers

Dr. Laureano Falla, Havana, Cuba and Dr. Jose R. Garmendia, Havana, Cuba (By invitation).

The following papers will be read by title and will appear in The American Journal of Gastroenterology.

1. "Hyperchlorhydria—Can it be Controlled Medically?"

Dr. Edwin Boros, New York, N. Y.

2. "Volvulus in the Newborn".

Dr. Roy Morrow, Jersey City, N. J. and Dr. Kenneth Judy, Jersey City, N. J.

VISIT THE EXHIBITS

3. "Incidence and Treatment of Endemic Fish Tapeworm Infection".
Dr. Milton J. Matzner, Brooklyn, N. Y., Dr. J. Rosenberg, Brooklyn, N. Y. and Dr. E. Neuman, Brooklyn, N. Y.

4. "A Consideration of Gastroduodenal Disorders".
Dr. Peter G. Brandetsas, Palmyra, N. Y.

5. "Carcinoma of the Gallbladder".
Dr. Edward F. Sciorsci, Hoboken, N. J.

6. "The Pancreatic Secretin Test as a Diagnostic Aid".
Dr. Dan Zavela, Detroit, Mich., Dr. Louis Guzzetta, Detroit, Mich. and Dr. Ruth Davis McNair, Detroit, Mich.

COURSE IN POSTGRADUATE GASTROENTEROLOGY

SURGICAL COORDINATOR AND CO-CHAIRMAN
OWEN H. WANGENSTEEN, B.A., M.D., Ph.D., Minneapolis, Minn.

MEDICAL COORDINATOR AND CO-CHAIRMAN
I. SNAPPER, M.D., Ph.D., Brooklyn N. Y.

FIRST SESSION

THURSDAY MORNING, 28 OCTOBER 1954

LYNN A. FERGUSON, M.D., F.A.C.G., President, American College of Gastroenterology, presiding.

9:00 A.M.
Address of Welcome —
DR. LYNN A. FERGUSON, Grand Rapids, Mich.

9:15 A.M.
1. "Hiatal Hernia, Achalasia and Epiphrenic Diverticulum".

Speaker
DR. F. J. PUTNEY, Philadelphia, Pa.

9:45 A.M.
2. "Conservative Management of Occlusive Diseases of the Esophagus".

Speaker
DR. KARVER L. PUESTOW, Madison, Wisc.

10:15 A.M. Recess to visit commercial, technical and scientific exhibits.

10:45 A.M.
3. "Hiatal Hernia".

Speaker
DR. BRIAN BLADES, Washington, D. C.

11:15 A.M.
4. "Hormonal Influences Upon the Stomach".

Speaker
DR. SEYMOUR J. GRAY, Boston, Mass.

VISIT THE EXHIBITS

SECOND SESSION

THURSDAY AFTERNOON, 28 OCTOBER 1954

2:00 P.M.

5. "Experimental Methods for the Evaluation of Therapeutic Agents in Peptic Ulcer".

Speaker

DR. FRANK P. BROOKS, Philadelphia, Pa.

2:30 P.M.

6. "The Present Status of Vagotomy in the Treatment of Duodenal Ulcer".

Speakers

DR. RALPH COLP, New York, N. Y. and DR. VERNON A. WEINSTEIN, New York, N. Y.

3:00 P.M. Recess to visit commercial, technical and scientific exhibits. (Exhibits close at 4:30 P.M.).

3:30 P.M.

7. "Management of Syndrome after Human Total Gastrectomy".

Speaker

DR. MOSES PAULSON, Baltimore, Md.

4:00 P.M.

8. "Gastric Lesions in the Aged, A Comparison of Surgically Treated Lesions in the Young and Old Age Group of Patients".

Speakers

DR. OTTO C. BRANTIGAN, Baltimore, Md. and DR. JOHN D. HAYNES, Baltimore, Md.

4:30 P.M.

9. "Prognosis and Treatment in Chronic Hepatitis".

Speaker

DR. W. PAUL HAVENS, JR., Philadelphia, Pa.

VISIT THE EXHIBITS

THIRD SESSION

FRIDAY MORNING, 29 OCTOBER 1954

All sessions on Friday will be held at Walter Reed Army Hospital, Washington, D. C. Bus transportation will be furnished and will leave from the main entrance of The Shoreham promptly at 8:30 A.M.

LT. COL. EDDY D. PALMER, M.C., Moderator.

9:00 A.M.
Address of Welcome —

MAJ. GEN. LEONARD A. HEATON, U.S.A., Commanding General.

9:05 A.M.
10. "The Roentgenological Manifestations of Esophagitis".

Speaker
COL. ELMER A. LODMELL, M.C.

9:35 A.M.
11. "The Esophagus During Pregnancy".

Speakers
MAJ. NORMAN M. SCOTT, JR., M.C. and LT. COL. DAVID L. DEUTSCH, M.C.

10:05 A.M.
12. "Hepatic Failure".

Speaker
CAPT. THOMAS C. CHALMERS, M.C.

10:35 A.M. Recess

10:45 A.M.

13. "Portal Decompression in Cirrhosis".

a. "Surgical Aspects".
LT. COL. CARL W. HUGHES, M.C. and MAJ. EDWARD J. JAHNKE, JR., M.C.

b. "Medical Aspects".
MAJ. IRVIN C. PLOUGH, M.C.

VISIT THE EXHIBITS

11:30 A.M.

LUNCHEON—at the Officers' Club. (admission by card only to be obtained at time of registration).

FOURTH SESSION

FRIDAY AFTERNOON, 29 OCTOBER 1954

1:00 P.M.

14. "PANEL DISCUSSION ON THE MANAGEMENT OF GASTRIC ULCER".

Moderator: Col. Frances W. Pruit, M.C.

Participants: Dr. Owen H. Wangensteen, Minneapolis, Minn.
Col. Robert T. Gants, M.C.
Lt. Col. Eddy D. Palmer, M.C.
Dr. Irving B. Brick, Washington, D. C.

2:30 P.M.

15. "Oral Lesions of Gastroenterologic Interest".

Speaker

Dr. Raymond A. Osbourn, Washington, D. C.

3:00 P.M. Recess

3:15 P.M.

16. "Bacteriology of Peritonitis".

Speaker

Maj. Henry H. Balch, Jr., M.C.

3:45 P.M.

17. "The Surgical Treatment of Diverticulitis Coli".

Speakers

Maj. Jack B. Jay, M.C. and Col. Robert T. Gants, M.C.

4:15 P.M. *Bus transportation back to The Shoreham will be furnished.*

VISIT THE EXHIBITS

FIFTH SESSION

SATURDAY MORNING, 30 OCTOBER 1954

This session and the final session in the afternoon will again be held at The Shoreham.

9:00 A.M.

18. "Diseases of the Pancreas".

Speaker

Dr. Samuel Morrison, Baltimore, Md.

9:30 A.M.

19. "Current Trends in the Management of Pancreatitis".

Speakers

Dr. Luther M. Keith, Jr., Columbus, Ohio and Dr. Joseph A. Bonta, Columbus, Ohio.

10:00 A.M. Recess

10:15 A.M.

20. "Unusual Problems in Biliary Tract Surgery".

Speaker

Dr. Robert J. Coffey, Washington D. C.

10:45 A.M.

21. "Present Day Management of Ulcerative Ileocolitis with Particular Reference to the Use of the Hormones and Newer Drugs".

Speaker

Dr. J. Arnold Bargen, Rochester, Minn.

11:15 A.M.

22. "The Elimination of Postoperative Morbidity in the Surgical Management of Chronic Idiopathic Ulcerative Colitis".

Speakers

Dr. Karl E. Karlson, Brooklyn, N. Y. and Dr. Clarence Dennis, Brooklyn, N. Y.

VISIT THE EXHIBITS

SIXTH SESSION

SATURDAY AFTERNOON, 30 OCTOBER 1954

2:00 P.M.

23. "Anomalies of Intestinal Rotation".

Speaker

DR. CLARENCE E. GARDNER, JR., Durham, N. C.

2:30 P.M.

24. "Growth-promoting Effects of Antibiotics".

Speaker

DR. CARL A. BAUMANN, Madison, Wisc.

3:00 P.M.

25. "Postneurotic Cirrhosis of the Liver".

Speaker

DR. DAVID M. SPAIN, Brooklyn, N. Y.

3:30 P.M.

26. "Rectal Problems".

Speaker

DR. WALTER H. GERWIG, JR., Washington, D.C.

SCIENTIFIC EXHIBITS

BOOTH S-1 "Peptic Ulcer, Treatment with Prantal"

Dr. Theodore S. Heineken, Bloomfield, N. J.

A review of over 100 peptic ulcer patients treated with a new cholinergic blocking agent, Prantal Methylsulfate, is presented. The outline of the treatment is given with the incident of side actions. The percentage of healing, as demonstrated by x-ray, is shown as well as the pain relief obtained. A graph of the reduction in gastric acidity is also shown when the drug is given intramuscularly. X-rays show the delay in emptying of the stomach before and after taking Prantal. Four complete case histories are presented (two of duodenal ulcers and two of gastric ulcers) with x-rays before and after treatment. From clinical experience of over three years, Prantal offers the physician a safe effective drug for therapy of peptic ulcers.

BOOTH S-2 "Roentgen Manifestations of Scleroderma"

Dr. Oliver S. Cramer, Albuquerque, N. M., Dr. J. W. Grossman, Albuquerque, N. M. and Dr. M. Stark, Albuquerque, N. M.

This exhibit consists of four panels of transparencies which show the changes seen in scleroderma (acrosclerosis) by printed description, photographs and x-ray copies which are reduced in size.

Panel I gives the general definition, classification, pathology and photographs of the face and hands of three patients.

Panel II shows the x-ray changes in the bones particularly the hands.
Panel III shows the changes seen in serial chest x-rays, consisting of increased markings and calcifications.

Panel IV shows the x-ray changes in the gastrointestinal tract. There are 5 x-rays showing typical changes in the esophagus, principally the dilated atonic esophagus and one stricture. There are 8 x-rays showing the typical roentgen findings in the stomach and small intestine. These consist of a dilated stomach and duodenum, and changes in the wall of the small intestine which are considered to be typical of this disease.

BOOTH S-3 "Basic Experiences in the Partially Collapsed Opaque Balloon Study of Small Bowel Activity"

Dr. Peter G. Brandetsas, Palmyra, N. Y. and Dr. A. Grohowski, Palmyra, N. Y.

This series of flat plate films of the abdomen show the passage of a partially collapsed balloon (containing a mixture of barium-water in ratio 1:4), through the small bowel. This allows a standard resistant force that lends itself to the contractile and propulsive forces of the small bowel and illustrates a normal standard that may form the basis for study of the pathologic bowel. Further refinements and development are expected to allow its application to clinical study of the small bowel, for motility, deficiency states, structural derangements (Meckel's diverticulum, bowel duplication), and small bowel tumors.

VISIT THE EXHIBITS

BOOTH S-4 **"Surgical Management of Ulcerative Colitis"**

DR. CHARLES B. RIPSTEIN, Brooklyn, N. Y., DR. F. SIEGMAN, Brooklyn, N. Y. and S. R. WAINE, Brooklyn, N. Y.

The exhibit demonstrates the rationale of colonic resection in ulcerative colitis. The type of pathology found, the complications, and the treatment of 150 cases are presented. Indications for operation are outlined, and the reasons for failure with less radical procedures are discussed.

BOOTH S-5 **"Topographic Anatomy of the Anorectal Canal"**

DR. EDWARD LEVY, Bronx, N. Y.

The exhibit consists of 10 pieces, – 9 illustrations and one set of legends. The illustrations are all originals. They consist of black and white photographs, with legends, of pelvic sections, made in the Anatomy Department of N. Y. U. Medical College. Line drawings, based on the photographs, were made by James Didusch, Staff Artist, Carnegie Institute of Embryology, Baltimore, Md. The illustrations depict the anorectal canal – its relations and directions. The topographic anatomy shown provides, among other facts of clinical significance, the anatomic basis for the atraumatic physical examination of the distal 25 cm. of the intestinal canal.

BOOTH S-6 **"Trigger Mechanisms in Abdominal Disturbances"**

DR. JACOB MELNICK, Brooklyn, N. Y.

The trigger mechanism is consistently present in abdominal disturbances. The component trigger areas are specific in relation to the physical findings and the trigger area patterns are constant for specific symptoms. The trigger mechanism persists after the original disturbance has subsided and may then become the major or even the sole cause of the persistence of the signs and symptoms. Illustrations and charts of the specific trigger areas and their reference zones are presented. Treatment is described and illustrated. The results of such treatment are presented in more than 100 patients with longstanding and intractable symptoms. The importance of the trigger mechanism in diagnosis is stressed.

TECHNICAL EXHIBITORS

(Those attending the Convention sessions are urged to take advantage of the time in between the presentation of papers and sessions, to visit the technical exhibits and become acquainted with the many new products and new equipment on display.)

AMES COMPANY, INC., Elkhart, Ind. (Booth 19). *Ictotest,* a 30-second accurate and simple tablet test for the detection of urine bilirubin as an aid to the diagnosis and management of jaundice and hepatitis, will be demonstrated. *Para Decholin, Decholin Sodium,* and *Decholin Belladonna,* adjuncts of choice in treating hepatobiliary disturbances, will also be on display.

THE BORDEN COMPANY, New York, N. Y. (Booth 25).
Mull-Soy, the original hypoallergenic soy food, will be on display with complete and up-to-date information on its use in dietary management of ulcers and for special dietary uses. Representatives will explain to you the extensive clinical background and rationale on time-proven *Mull-Soy.* Reprints and samples are available to you.

VISIT THE EXHIBITS

BRISTOL LABORATORIES, INC. New York, N. Y. (Booth 10). A replica of an old-time apothecary shop will feature antibiotic products and pharmaceutical specialties. Qualified representatives will be on hand to supply you with information on the Bristol line including *Centrine Tablets, Centrine Tablets* with Phenobarbital, *Centrine Solution, Alminate Tablets, Barbonate Tablets* and *Kectil.*

BURTON, PARSONS & COMPANY, Washington, D. C. (Booth 23), will feature their hydrophilic colloids, *Konsyl* and *L. A. Formula. Konsyl* is the sugar-free hydrophilic colloid. It contains 100 per cent bulk-producing material and contains no sugars, binders or other diluents. Because *Konsyl* has no caloric value it is ideal for use with the diabetic, the obese and others with restricted caloric intake – as well as your routine constipation cases.

L. A. Formula is the hydrophilic colloid unsurpassed for palatability. It literally defies detection in orange juice or milk. Moreover, *L. A. Formula* takes up to 10 minutes to gel in these vehicles, allowing even your slowest patient sufficient time to drink this smooth mixture.

Konsyl and *L. A. Formula* are the most economical bulk producers available on the market today.

CIBA PHARMACEUTICAL PRODUCTS, INC., Summit, N. J. (Booth 7), will feature *Antrenyl,* a new potent anticholinergic agent with no bitter after-taste in the management of peptic ulcer and spasm of the gastrointestinal tract. Samples and literature will be available.

COCA-COLA LOUNGE — Ice cold Coca-Cola served through the courtesy and cooperation of the Washington Coca-Cola Bottling Works and the Coca-Cola Company.

DOHO CHEMICAL CORPORATION, New York, N. Y. (Booth 12), are pleased to exhibit the following:

Auralgan, the ear medication for the relief of pain in Otitis Media and removal of Cerumen; *New Otosmosan,* the effective, non-toxic ear medication which is Fungicidal and Bactericidal (gram negative-gram positive) in the suppurative and aural dermatomycotic ears:

Rhinalgan, the nasal decongestant which is free from systemic or circulatory effect and equally safe to use on infants as well as the aged.
Mallon Chemical Corp., subsidiary of the Doho Chemical Corp. will also feature:

Rectalgan, the liquid topical anesthesia, also for relief of pain and discomfiture in hemorrhoids, pruritus and perineal suturing.

EDER INSTRUMENT CO., Chicago, Ill. (Booth 5), will again exhibit their latest developments in gastroscopic equipment. Several instruments with considerable improvements will be introduced other diagnostic instruments will also be exhibited.

P. LORILLARD CO., NEW YORK, N. Y. (Booth 18). An interesting demonstration of the advantages of the exclusive Micronite® filter used in Kent cigarettes. Representatives will be present to demonstrate and answer questions.

THE NATIONAL DRUG COMPANY, Philadelphia, Pa. (Booths 26 and 27).

THE O-CEL-O Division of GENERAL MILLS, Buffalo, N. Y. (Booth 14), will exhibit the new surgical sponge, *Cel-O-Sorb.* A colored film will run continuously showing these sponges in use in surgery. The film also illustrates other advantages claimed, including economy of time, money, space and labor. Sample requests will be accepted.

VISIT THE EXHIBITS

ORGANON INC., Orange, N. J. (Booth 17), will feature a new development in the antacid field – *Trevidal®. Trevidal* is the first antacid to offer four effective antacid ingredients carefully and clinically balanced to avoid constipation or diarrhea, plus *Regonol* (a new and unique vegetable mucin which provides a protective coating even in a highly acid environment), and *Egraine,* a protein binder from oat which prolongs and controls antacid activity. Visit the Organon booth for literature and samples.

PET MILK COMPANY, St. Louis, Mo. (Booth 22), will be pleased to have you stop and taste the "fresh milk flavor" of *Pet Nonfat Dry Milk.* Our representative will be on hand to serve you and discuss the use of *Pet Nonfat Dry Milk* in special diets.

J. B. ROERIG AND COMPANY, Chicago, Ill. (Booth 21). Information, Sample and literature will be available on *Tetracyn* – the newest broad spectrum antibiotic, also our well known nutritional products, such as *Viterra, Viterra Therapeutic, Heptuna Plus, Amplus, Obron, Obron Hematinic,* etc.

WILLIAM H. RORER, INC., Philadelphia, Pa. (Booth 15), will feature *Probutylin* – a new drug which provides a new approach to treatment of nausea, vomiting, pylorospasm, etc. *Probutylin* has been clinically tested over a three year period, and case reports will be available at their booth for inspection. Suspension *Maalox* and Tablets *Maalox,* the nonconstipating, Magnesium Hydroxide-Aluminum Hydroxide Colloidal Gel, useful in Peptic Ulcer, Gastritis and Heartburn associated with Pregnancy, will also be presented.

SANDOZ PHARMACEUTICALS, Hanover, N. J. (Booth 9), cordially invites you to visit our display at the National Gastroenterological Association Meeting. They plan to feature new information on certain established products and complete data on *Plexonal*–the newest member in their family of ethical specialties.

Cafergot–Available in oral and rectal form for effective control of head pain in migraine and other vascular headaches.

Bellergal–A time-tested preparation for use in functional disorders.

Plexonal–A new sedative-hypnotic – *Plexonal.* This exhibit demonstrates that *Plexonal* is not just another sedative, but is one developed on a new pharmacologic approach. The action of subthreshold doses of classic sedative agents are potentiated and enhanced by autonomic and central acting drugs.

Belladenal–Sandoz presents an exhibit on *Belladenal* an antispasmodic sedative for the control of hypermotility with pain and of hypersecretion of the intestinal tract.

SCHENLEY LABORATORIES, INC., New York, N. Y. (Booth 16), features:

Ediol–A Palatable oral fat emulsion for quick gain in weight and energy.

Titralac–An effective antiacid because it titrates like milk.

Sedamyl–A non-barbiturate, ideal for daytime sedation without drowsiness.

Dorbane–A precise potency for individualized dosage in the treatment of constipation.

SCHERING CORPORATION, Bloomfield, N. J. (Booth 8). Members of the National Gastroenterological Association and their guests are cordially invited to visit the Schering exhibit where new therapeutic developments will be featured. Their representatives will be present to welcome you and to discuss with you these products of their manufacture.

G. D. SEARLE & CO., Chicago, Ill. (Booth 6). You are cordially invited to visit the Searle booth where their representatives will be happy to answer any questions regarding Searle Products of Research.

VISIT THE EXHIBITS

Featured will be *Vallestril,* the new synthetic estrogen with extremely low incidence of side reactions; *Banthine,* and *Pro-Banthine,* the standards in anticholinergic therapy; and *Dramamine,* for the prevention and treatment of motion sickness and other nauseas.

E. R. SQUIBB AND SONS, New York, N. Y. (Booth 11).

WINTHROP-STEARNS INC., New York, N. Y. (Booth 24), extends a cordial invitation to visit their booths. Featured will be:

Monodral, a new, well tolerated, synthetic anticholinergic for peptic ulcer (gastric or duodenal), hyperacidity, pylorospasm, spastic and irritable colon. *Monodral* has unusual gastric antisecretory power, producing temporary antacidity in many patients.

Creamalin, nonalkaline, nonabsorbable antacid.

WYETH LABORATORIES, Philadelphia, Pa. (Booth 4), will feature *Amphojel®,* aluminum hydroxide gel and *Aludrox®,* aluminum hydroxide with magnesium hydroxide, two widely prescribed antacids, for rapid relief of simple hyperacidity and for medical management of peptic ulcer. Also featured will be *Streptomagma®,* palatable suspension of dihydrostreptomycin, pectin and kaolin in alumina gel, for prompt and complete remission of many bacterial diarrheas.

President's Message

There exists in the minds of many of our Fellows and Members confusion regarding the recent activation of the American College of Gastroenterology. This is natural since most of our members have not had the opportunity of following the progress of the actual procedures involved, and of course, most of the details have had to be worked out in committee meetings.

Restraint and failure to criticize have been predominant during this formative period. For this your officers have been grateful and most appreciative.

As we approach the actual operation of our College, many questions and problems will arise. These are now in order. If our new organization is to function properly, then these matters concern the Fellowship, and it will be their opportunity to bring them up for discussion at the annual meeting. There, by vote of the majority of Fellows, action will be taken.

The College has vested all authority in the Fellowship and entrusted the carrying out of its mandates to a Board of Trustees and an Executive Committee. A new Board of Governors has been created as a liaison group between the Board of Trustees and the Fellowship. This arrangement permits a much greater participation by our Fellows and Members in the conduct and operation of the College than was true in our former organization. Now the opportunity will be available for the Fellows to actively participate and we hope they will do so.

Furthermore, we now have in one organization fellowship with all physicians interested in gastroenterology and this should prove most helpful in studying and solving many problems in this field. Fellowship is on one basis and that is adequate training in each of the fields touching on gastroenterology, namely: Internal Medicine, Surgery, Radiology, Proctology and Pathology.

It is hazardous to make prophecies, but I have an unbounded confidence that a great future lies before us. Each one of us can have a hand in shaping this future by taking an active part in all the activities offered by the College.

Sigurd W. Johnsen

EDITORIAL

Prolapse of the Gastric Mucosa Through the Pylorus into the Duodenum

It is only within recent years that prolapse of the gastric mucosa through the pylorus into the duodenum has been recognized as a diagnostic and therapeutic problem. Feldman and Myers discuss this condition and its recognition by gastrointestinal roentgen examination, claiming that in 371 patients they found 14 per cent of prolapse of the gastric mucosa. These figures are much higher than 0.1 to 7.7 per cent found in surveying the literature. Many patients were also found to have ulcers in addition to the prolapse.

It is interesting to note that in all of these patients the etiological factors were a redundant gastric mucosa, gastritis with hypertrophy of the mucosal folds and heightened peristaltic activity. Idiopathic gastric hemorrhage or persistent occult bleeding without ulcer may be caused by prolapse of the gastric mucosa and its differentiation from ulcer can only be made by careful roentgen study.

Clinically, prolapse of the gastric mucosa is a medical problem and surgery is indicated only in the intractable cases, or where the large prolapse becomes incarcerated in the pylorus causing stenosis or bleeding.

In treating prolapse of the mucosa it is advisable to overcome the marked peristaltic activity which is responsible for the propulsion and extent of the prolapse of the gastric mucosa, by giving the patient cholinergic and ganglionic blocking agents to suppress or abolish gastric peristalsis. Stoppage of peristaltic activity will act as a physiologic test in reducing or eliminating the prolapse, and will further aid as a diagnostic and therapeutic measure for the treatment of this condition.

When the physician cannot find a definite cause for the patient's complaint, he should think of the possibility of prolapse or herniation of the gastric mucosa. When roentgenography is not available or is refused by the patient, prescribing cholinergic and ganglionic blocking agents and a bland diet, may often relieve the patient of his symptoms. Failure to be relieved requires further investigation.

Samuel Weiss, M.D.

NEWS NOTES

Annual Convention

The Convention in Washington, D. C., to be held at The Shoreham, 25,26, 27 October 1954 will be the Nineteenth and Final Convention of the National Gastroenterological Association. This will also be the First Annual Convention of the recently activated American College of Gastroenterology.

Copies of the program for the Convention are being mailed separately to the membership. Additional copies are available from the Headquarters office, 33 West 60th St., New York 23, N. Y. They will also be available at the registration desk on the Convention floor.

In addition to various individual papers, the program will include panel discussions on "Twenty-five Years' Observation of the Gallbladder Controversy" and "Amebiasis", the latter to be given by the staff of the National Institutes of Health in Bethesda, Md. There will also be a symposium on "Esophageal Varices".

The Wednesday evening session, which has in previous years proved to be quite successful, will again be included.

Ladies Auxiliary Program

An interesting and entertaining program has been planned for the ladies attending the Washington, D. C. Convention, by Mrs. Sigurd W. Johnsen of Upper Montclair, N. J., President of the Ladies Auxiliary, and her committee. Letters will be sent to the ladies with a business reply card asking them to make their reservations for each of the activities.

The program for the ladies follows:

Monday, 25 October 1954

Registration at the Registration desk in the hotel from 8:30 A.M.

Business meeting of the Auxiliary at 5:00 P.M.

Convocation Ceremony, 6:30 P.M.

President's Annual Reception, 8:00 P.M.

(*Tickets for the President's Reception will be given out only to those attending the Convocation Ceremony.*)

Tuesday, 26 October 1954

Sightseeing tour of Washington, Arlington National Cemetery and Mt. Vernon with a special stop to visit the White House, leaving the hotel 9:30 A.M.

Annual Banquet, 7:00 P.M. (to be followed by dancing).

Wednesday, 27 October 1954

Luncheon at The Shoreham, 12 Noon.

Television show at the Sheraton-Park Hotel, 2:00 P.M.

Tentatively a tea at one of the Foreign Embassies if this can be arranged.

Thursday, 28 October 1954

Postconvention tour of the Federal Bureau of Investigation, 2:00 P.M.

The cooperation of the ladies in returning the postcards immediately will aid the committee in making arrangements for these activities.

Registration

Registration for the Convention will take place on the Convention floor at The Shoreham. Those attending are requested to register and receive their identification badges as no one will be admitted to the exhibits or sessions without a badge.

Scientific Exhibits

Scientific exhibits, which again are a part of the Convention program, will be in the exhibit hall and will be on display from Monday afternoon through Thursday afternoon. Those exhibiting have been asked to have a representative present at their booth before and after each session and during recess.

Coffee Hour

On Tuesday and Wednesday mornings, 26 and 27 October, coffee and doughnuts will be served in the exhibit area from 8:30 A.M. to 9:00 A.M. This innovation will be with the compliments of the Association. Those attending the Convention are invited to avail themselves of this service.

Board of Trustees

The Annual Meeting of the Board of Trustees will be held at The Shoreham in Washington, D. C. at 4:00 P.M. on Sunday, 24 October 1954. Following the meeting there will be a banquet for the officers and the board.

Annual Meeting of the American College of Gastroenterology

The Annual Meeting of the American College of Gastroenterology will be held at The Shoreham in Washington, D. C., at 5:00 P.M. on Monday, 25 October 1954.

Fellows of the College are requested to attend and participate in the business sessions. Election of officers and Board of Governors will be held at that time.

Annual Meeting of the National Gastroenterological Association

The Annual Meeting of the National Gastroenterological Association will be held at The Shoreham in Washington, D. C. at 6:00 P.M. on Monday, 25 October 1954.

This will be the final meeting of the Association.

Convocation Ceremony

The Convocation Ceremony, at which certificates will be presented in person to newly elected Members, Associate Fellows, Fellows and those advanced during the past year, will follow the Annual Meetings at 6:30 P.M. on Monday evening, 25 October 1954 at The Shoreham, in Washington, D. C.

The principal speaker will be Major General Silas B. Hays, Deputy Surgeon General of the Army.

Members, their families, guests and friends are invited to attend.

President's Annual Reception

The President's Annual Reception, again to be sponsored by Winthrop-Stearns, Inc., will be held at 8:00 P.M. on Monday evening, 25 October 1954, at The Shoreham in Washington, D. C.

Members of the Association, their friends, guests and those attending the Convention are cordially invited to attend.

Admission cards may be obtained *only* at the Convocation Ceremony which precedes the reception.

Annual Luncheon

Commencing with the Convention this year, an Annual Luncheon for those attending the sessions will be sponsored by Burton, Parsons & Co. The luncheon will be held on Tuesday, 26 October 1954 in the West Ball Room of The Shoreham in Washington, D. C.

A prominent speaker will deliver a short address on a timely topic of medical interest. Tickets of admission may be obtained at the registration desk on the Convention floor.

Organizational Meeting of the Board of Governors

The organizational meeting of the Board of Governors of the American College of Gastroenterology will be held at The Shoreham in Washington, D. C. on Tuesday, 26 October 1954 at 5:00 P.M.

Election of a chairman and appointment of committees will take place at this time.

Annual Banquet

The Annual Banquet of the National Gastroenterological Association will be held at The Shoreham in Washington, D. C. on Tuesday evening, 26 October 1954 at 7:00 P.M.

The incoming President, Dr. Lynn A. Ferguson of Grand Rapids, Mich. will be formally installed at that time.

There will be a principal speaker and the banquet will be followed by dancing.

Tickets at $7.50 per person will be available at the registration desk on the Convention floor.

All reservations must be made by 10:00 A.M., Tuesday, 26 October 1954.

Course in Postgraduate Gastroenterology

The Sixth Annual Course in Postgraduate Gastroenterology, given by the National Gastroenterological Association, will be held at The Shoreham and Walter Reed Army Hospital in Washington, D. C. on 28, 29, 30 October 1954.

The two coordinators of the course, Dr. Owen H. Wangensteen, Professor and Chairman of the Department of Surgery, University of Minnesota Medical School and Dr. I. Snapper, Director of Medical Education, Beth-El Hospital, Brooklyn, N. Y. will again personally direct the sessions.

In addition to Drs. Wangensteen and Snapper, a distinguished faculty chosen from various medical schools and the Walter Reed Army Hospital Staff will present the course.

Admission to the course sessions will be limited to those who hold matriculation cards indicating that they have paid their fee for the course.

Nominating Committee Report

The Nominating Committee of the American College of Gastroenterology, consisting of Dr. Sigurd W. Johnsen, Passaic, N. J., Chairman; Dr. Lynn A. Ferguson, Grand Rapids, Mich.; Dr. William E. Bippus, West Palm Beach, Fla.; Dr. Harry Barowsky, New York, N. Y. and Dr. Michael W. Shutkin, Milwaukee, Wisc., has unanimously submitted the following slate of candidates to be voted upon at the Annual Meeting of the College in October:

Officers

President-Elect..... James T. Nix, M.D., New Orleans, La.
1st Vice-President..... Arthur A. Kirchner, M.D., Los Angeles, Calif.
2nd Vice-President..... C. Wilmer Wirts, M.D., Philadelphia, Pa.
3rd Vice-President..... Frank Borrelli, M.D., New York, N. Y.
4th Vice-President..... Fred H. Voss, M.D., Phoenicia, N. Y.
Secretary..... Joseph Shaiken, M.D., Milwaukee, Wisc.

Board of Trustees

For 1 Year: To fill the unexpired term, ending in 1955, of Dr. Ludwig Frank who resigned— William W. Lermann, M.D., Pittsburgh, Pa.

Board of Governors

California..... Lester M. Morrison, M.D., Los Angeles
Connecticut..... Max Caplan, M.D., Meriden
Florida..... Marvin Smith, M.D., Miami
Illinois..... Edward J. Krol, M.D., Chicago
Louisiana..... Louis Ochs, Jr., M.D., New Orleans
Massachusetts..... Henry Baker, M.D., Boston
Michigan..... James A. Ferguson, M.D., Grand Rapids
New Jersey..... Theodore S. Heineken, M.D., Bloomfield
New York City..... Harry Barowsky, M.D., New York
New York State excluding
New York City..... Libby Pulsifer, M.D., Rochester
Ohio..... Edward A. Marshall, M.D., Cleveland
Pennsylvania..... N. Keith Hammond, M.D., Pittsburgh
Rhode Island..... Irving Beck, M.D., Providence
Tennessee..... E. G. Campbell, M.D., Memphis
Wisconsin..... Robert T. McCarty, M.D., Milwaukee

American College of Physicians To Use Nationwide TV Closed Circuit Telecast in Connection With Its Postgraduate Program

On Thursday evening, 23 September 1954, from 6:00 P.M. to 7:00 P.M., Eastern Daylight Saving Time, the American College of Physicians will utilize

television through a national closed circuit over the Columbia Broadcasting System to carry to its members and their colleagues a *Symposium On The Management Of Hypertension*. This telecast is made possible through the co-operation and generous support of Wyeth Incorporated of Philadelphia, and will be the *first nationwide* closed circuit hookup for postgraduate medical education.

The panel of distinguished physicians who will participate includes: Cyrus C. Sturgis, M.D., F.A.C.P., Presiding, President, American College of Physicians, Professor of Internal Medicine, University of Michigan, Ann Arbor; F. H. Smirk, M.D., F.R.A.C.P., Professor of Medicine, University of Otago, Dunedin, New Zealand; R. W. Wilkins, M.D., F.A.C.P., Chief, Hypertension Clinic, Massachusetts Memorial Hospital, Boston; Garfield G. Duncan, M.D., F.A.C.P., Director of the Medical Division, Pennsylvania Hospital, Philadelphia and Edward D. Freis, M.D. (Associate), Adjunct Clinical Professor of Medicine, Georgetown University, Washington, D. C.

A "closed TV circuit" is one by which reception is controlled and not open to the general TV public. This telecast cannot be picked up in the home, but the invited audience must go to the TV receiving station. Twenty-three such receiving stations will be used; these will be located in Boston, New York, Philadelphia, Washington, Pittsburgh, Charlotte, Atlanta, Cincinnati, Detroit, Chicago, St. Louis, Milwaukee, Minneapolis, Memphis, Dallas, Houston, New Orleans, Denver, Salt Lake City, Los Angeles, San Francisco, Baltimore and Cleveland.

American Board of Internal Medicine

The next written examination of the American Board of Internal Medicine will be given on Monday, 18 October 1954.

Further information may be obtained by writing to Dr. William A. Werrell, Executive Secretary-Treasurer, 1 West Main St., Madison 3, Wisc.

In Memoriam

We record with profound sorrow the passing of Dr. John H. Alexander, Pittsburgh, Pa.; Dr. Walter Freymann, West New York, N. J., Fellow; Dr. Henry A. Rafsky, New York, N. Y., Fellow.

We extend our deepest sympathies to the bereaved families.

Hyman I. Goldstein, M.D.

Unable to attend the funeral services of our departed colleague, I am honored and comforted by our President's request that I deliver this brief eulogy*.

Concerning Hyman Goldstein's contributions to medicine, the record can hardly be improved upon tonight. Medical literature has already recorded his early work on "Hereditary, Hemorrhagic Telengiectasia" and reference to any modern, complete medical dictionary already contains the clinical entities of "Goldstein's disease," "Goldstein's Hematemesis" and "Goldstein's Hemoptysis".

The next subject to engage his interest was the study of malignant neoplasms of the gastrointestinal tract and liver, particularly sarcoma.

Finally, as is so well known to you he devoted himself to medical history in which field he became an outstanding authority.

During the first 34 years of his professional life he contributed about 125 articles to the medical literature†.

I would, however, like to dwell upon three different aspects of his career, namely:— 1. The rich life of Hyman I. Goldstein. 2. His dynamic devotion to this Society. 3. The fitting manner of his death.

Concerning his rich life, those of you with a biblical turn of mind will recall with interest that a literal translation of his biblical first name means life. It is perfectly proper to refer to Hyman Goldstein in biblical terms because in spite of his many activities, he regularly attended the Friday evening bible classes until his death. The bible defines a rich man as one who is satisfied with his lot in life.

I had the privilege of visiting Hyman Goldstein at his office in Camden, N. J. and was impressed with the fact that he had the largest medical library of any physician I know. There were no elaborate mechanical gadgets conducive to a fashionable, lucrative practice.

*Delivered at the Annual Dinner of the New Jersey Gastroenterological Society at Newark, N. J., 14 April 1954.

†Kagan, S.A.J.: Internat. Coll. Surg. **10**:710, (Dec.), 1947.

He chose instead to travel extensively in the service of medicine. Thus, in his lifetime he crossed the ocean seven times and spent three full years of his professional life in the well known European medical centers, particularly Vienna.

As for being satisfied with his lot, you well remember his joviality, his humor, his chiding and above all the triumphant chuckle betraying a sense of complete inner satisfaction whenever he scored a point either in Internal Medicine or Medical History.

So far as his devotion to this Society is concerned, it can be stated for the record that he became a member of our organization in 1934 and was made an honorary life member in 1951; but this would not convey very much.

My mind goes back about 12 years to a meeting held at the home of Dr. Louis Perkel in Jersey City during a blizzard. While most of the members, residing within a radius of 5-7 miles from Jersey City, could not brave the elements, whom do you suppose was the first arrival?— Hyman I. Goldstein from Camden, N. J.

Most of you will also remember a meeting of our Society about two years ago at which a message from him was read stating that he was sorry to be unable to be present, as he was a patient at the University Hospital having just undergone an abdominoperineal resection.

This brings me to the third and final aspect of his career, namely, the fitting and symbolic manner of his death.

Following a double assault on his heart and intestinal tract, either one of which might have frozen some of us into idleness, he hit the trail again with renewed vigor, devoting the twilight of his life to his well known pet subject "Errors of Priority Credit in Medicine", a topic upon which he addressed the New England Society of Medical History about one year ago.

In the forenoon of March 17, 1954, while attending a seminar at the University of Pennsylvania Hospital on the topic of values to be derived from liver biopsy, as usual he was called upon to discuss the historical aspects.

I shall now give you the last words uttered by him: "Liver biopsies were done as long ago as 1840. It is regrettable that those who have resurrected the procedure failed to give priority credit". With this statement he collapsed and within a matter of minutes he was dead.

In memory of this brilliant clinician, distinguished medical historian and devoted member of our Society, Hyman I. Goldstein, born in Baltimore, Md., on November 2, 1887, died in Philadelphia, Pa., March 17, 1954, may I ask you to please rise for one minute of silent meditation.

I. GELBER

ABSTRACTS FOR GASTROENTEROLOGISTS

GASTROINTESTINAL TRACT

THE MECHANISM OF CARDIOSPASM: John Tilden Howard. Southern M. J., 46:729, (Aug.), 1953.

Cardiospasm must not be confused with diffuse spasm of the lower esophagus and with spasm of the cardia. The latter two entities denote irritability of the lower esophagus or the cardia respectively without any dilatation of the gullet above the spasm. True cardiospasm shows a dilated esophagus above the constricted segment. This narrowed area does not disappear even at the time of the operation with the patient under general anesthesia. No cause of this narrowing, however, can be found at the operating table. Careful microscopic examinations have revealed a disappearance of the ganglion cells of the myenteric plexus of Auerbach in the narrow, as well as in the dilated, portion. Balloon studies of the cardiospastic gullet have shown a reduced tone and an irregular activity of the intrinsic muscles leading to improper propulsion. Both findings indicate a damaged parasympathetic innervation which is also demonstrated by the extreme sensitivity of the cardiospastic esophagus to Mecholyl. There is considerable evidence that cardiospasm starts frequenlty after emotional stress, however psychiatric treatment alone is of very little benefit. It might be helpful as an adjunct in cases where dilatations are not completely successful.

H. B. EISENSTADT

GASTROINTESTINAL HEMORRHAGE AS A COMPLICATION OF POLIOMYELITIS: Robert J. Hoxsey. A. M. A. Arch. Int. Med., 29:662, (Nov.), 1953.

The histories of five fatal cases of poliomyelitis are added to several others previously reported who showed gastrointestinal hemorrhage as a terminal complication. Hemorrhagic gastritis, gastric erosions and acute esophageal, gastric and duodenal ulcers were found to have caused the bleeding in these cases where an autopsy was performed.

Trauma of suction tubes and mechanical respirator, anoxia and vitamin and food deficiencies were thought to be contributing factors while the main cause seemed to be midbrain lesions producing Cushing-like ulcerations. Gastrointestinal bleeding occuring during poliomyelitis carries a serious prognosis. Only one of the reported cases has survived. Treatment consists of a Sippy regimen in addition to blood transfusions.

H. B. EISENSTADT

A CLINICAL REPORT ON THE CYTOLOGIC DIAGNOSIS OF GASTRIC CANCER: O. F. Gurves, H. F. Traut, D. A. Wood and S. M. Farber. Surg. Gynec. & Obst. pp. 347-352, (March), 1954.

A brief report of the contribution of papain lavage technic to the effectiveness of gastric cytology is presented and four cases studied by this method are described. In three of these cases, cytologic study demonstrated the presence of cancer after all

other diagnostic methods had failed. Resection was possible in each instance and the patients are apparently free of disease to date, from 3 years to 42 months after operation. These cases emphasize the value of cytology in detecting malignant lesions of the stomach while they are still not only resectable but in a stage in which surgical treatment may affect long term cures, rather than merely palliative results. It is suggested that a wider use of cytology as a routine method of evaluation of gastric symptoms can make a substantial contribution to the control of malignant lesions. Its use is mandatory, the authors believe, in the diagnosis of any gastric complaint in patients over 50 years of age.

J. R. Van Dyne

THE EFFECTS OF MASSIVE GASTROINTESTINAL HEMORRHAGE ON HEMOSTASIS; I-THE BLOOD PLATELETS: J. F. Desforges, F. S. Bigelow and T. C. Chalmers. J. Lab. & Clin. Med. pp. 501-510, (April), 1954.

Significant changes were observed in the platelet count during and after massive gastrointestinal bleeding in patients with and without liver disease. There was a lowering of the count during and for several days after bleeding, followed by a definite rise with convalescence. The decrease in platelet count was not related to the amount of blood loss or to the presence or absence of shock. There was no evidence that the occasional marked drop in platelets contributed to the continuation of the hemorrhage. It is concluded from these studies that during gastrointestinal hemorrhage the platelets remain adequate to play their part in hemostasis along with the vascular system, both in normal patients and in those with severe liver disease, even though the platelet level may be extremely low.

J. R. Van Dyne

THE EFFECTS OF MASSIVE GASTROINTESTINAL HEMORRHAGE ON HEMOSTASIS; II-COAGULATION FACTORS: T. C. Chalmers, F. S. Bigelow and J. F. Desforges. J. Lab. Clin. Med. pp. 511-517, (April), 1954.

The observations of coagulation are similar to those of platelets in the preceding paper in that there was no evidence that any defect led to the continued bleeding. The tests failed to reveal any need for special types of replacement therapy other than the use of blood transfusions to maintain adequate hematocrit levels. They do not, however, exclude the possibility that more striking coagulation defects may appear in patients with more massive hemorrhage, treated less promptly. It was found in this study, that abnormalities were more frequent in cirrhotics in over all tests of clotting and in those designed to measure factors contributing to thrombin formation and fibrin disposition.

J. R. Van Dyne

OCCURRENCE OF ASCITES IN MYXEDEMA: Fred Madenberg, George V. Byfield and Lyle A. Baker. A. M. A. Arch. Int. Med., 93:787, (May), 1954.

Only a few cases of ascites due to myxedema have been reported in the literature. This complication may be a symptom of myxedema *per se* just like the periorbital and pretibial edema and the pleural and pericardial effusions. The cause of these changes is supposed to be an alteration of the body proteins. Quite often, however, ascites is a part of the congestive heart failure due to myocardial degeneration of myxedema or of coronary artery disease which is so frequently observed in connection with myxedema. Differentiation is difficult. Normal venous pressure and ascitic fluid high in protein and specific gravity, favor the first mechanism, while venous congestion and ascites with low protein and specific gravity (transsudate) speak for congestive failure.

Treatment has to be extremely careful in either condition; small doses of desiccated thyroid are specific for a primary myxedematous fluid accumulation. Digitalis, diuretics, and salt restriction are indicated in congestive failure.

H. B. Eisenstadt

STOMACH

THE TREATMENT OF GASTRODUODENAL ULCERS BY RADIOTHERAPY: M. Grunberg. Quart. Rev. of the Harefuah, J. M. A. Israel, 1-4:17-20, 1953. Proceedings of the Second World Congress of Jewish Physicians.

The development of radiotherapy is reviewed, with mention of the various contributors.

During the last 30 years, the classic therapy of gastric and duodenal ulcers with antacids and surgery has given disappointing results. After 1944, Dragstedt's operation of vagotomy raised high hopes, but has now been abandoned in Europe, and is now performed only after gastroenterostomy and secondary gastrectomy fail to effect a cure.

Regardless of the theories as to its etiology, gastric ulcer seems to be caused by an excess of gastric juice. While x-ray therapy has not reached its rightful place in our struggle against gastric ulcers, the excess of gastric juice is diminished or disappears after radiation.

Review of several series of cases from various authors, including 80 of the speaker's own, which totaled almost 300 cases, gave a total of over 90 per cent of cures or improvements, with the former predominating by a large margin.

ARNOLD L. BERGER

SARCOID AND SARCOID-LIKE GRANULOMAS OF THE STOMACH: Norman M. Scott Jr., Vernon M. Smith, Philip A. Cox and Eddy D. Palmer. A.M.A. Arch. Int. Med. 92:741, (Nov.), 1953.

Sarcoid and sarcoid-like granulomas of the stomach have been rarely reported in the literature mainly because they do not produce a uniform clinical syndrome. Such lesions have been accidentally found in stomachs resected for gastric and duodenal ulcer and gastric carcinoma. They have been described in connection with systemic histoplasmosis. Sarcoid and sarcoid-like granulomas may produce the pictures of pylorostenosis, linitis plastica, hemorrhagic gastritis and nonspecific dyspepsia. They may cause vague abdominal pains or may be completely asymptomatic. Gastric acidity is usually low or absent in the presence of such pathology. Two patients are reported: In one case the diagnosis of sarcoid was made by biopsy of the enlarged lacrimal glands. Here the involvement of the stomach was proven by vacuum tube biopsy. In the other case, intractable anorexia, vomiting, weight loss and hematemesis lead to surgical exploration, vagotomy and gastroenterostomy. Here the sarcoid lesions were found through surgical biopsy.

The differentiation of sarcoid and sarcoid-like granulomas from the socalled nonspecific granulomas of the stomach is not possible at this time.

H. B. EISENSTADT

BENIGN TUMORS OF THE STOMACH: H. Kook, R. Colp, and V. Weinstein, Harefuah. J. M. A. Israel. 45:223-224, (Dec. 1), 1953.

Three cases of benign mesenchymal tumors of the stomach, which were diagnosed preoperatively, are presented and discussed. Histological examination revealed that one of the three was malignant. The importance of radiography as the best diagnostic means is stressed as well as the advisability of greater frequency of its utlization. Recommended therapy is resection including a wide margin of apparently normal tissue, with immediate frozen section.

ARNOLD L. BERGER

VAGOTOMY FOR DUODENAL ULCER: John R. Brooks and Francis D. Moore. New England J. Med. 249: No. 27, (Dec. 31), 1953.

A large group of patients with duodenal ulcer treated by vagotomy alone or combined with posterior gastroenterotomy were reviewed at the end of a 10-year follow-up. The results of this form of treatment was evaluated in the light of clinical response and changes in physiological response in the stomach and duodenum as indicated by secretory tests and gastric motility. On the basis of the data gathered, the authors were able to conclude that vagotomy alone no longer has a place in the treatment of uncomplicated intractable duodenal ulcer, primarily because it failed to prevent recurrent ulceration and because subtotal gastric resection presented a much greater

promise of success. Also, it was noted that recurrent ulceration frequently occurred while the physiologic effects of vagotomy persisted. Vagotomy in the treatment of marginal ulceration following subtotal gastric resection is advocated.

WILLIAM E. JONES

PEPTIC ULCER: David T. Monahan. New England J. Med. 249:1012. (Dec. 17), 1953.

In the five-year period from 1947 through 1951 there were 146 operations as definitive treatment of ulcer, exclusive of those for perforation, carried out at the Bridgeport hospital. Resection was done in 109 cases, gastroenterotomy and vagotomy in 19; gastroenterotomy alone in 12 and vagotomy alone in 6. There were four deaths with an overall mortality rate of 2.7 per cent. The mortality rate in gastric resection only was 3.6 per cent, including those cases of massive bleeding and marginal ulcer. Reference is made to Moore's recent study of 738 medically treated ulcers with a mortality of 2.03 per cent and 175 cases of ulcer treated by gastric resection with a mortality of 2.86 per cent.

Thirty-two cases of resection were followed closely by the author. All of these patients survived and returned to their usual occupation in a matter of a few weeks to a month or so. Only one of the 32 patients did not feel symptomatically improved. About one-third of the patients remained at about 10-15 pounds less than their preoperative normal weight but all were considered well nourished.

Again, the advisability of gastric resection in chronic duodenal ulcer as a prevention of both complications and disability is considered.

WILLIAM E. JONES

MALLORY-WEISS SYNDROME: John P. Decker, Norman Zamcheck and G. Kenneth Mallory. New England J. Med. 249: No. 24, (Dec. 10), 1953.

Clinical and pathological observations are given on 11 cases of hemorrhage from gastroesophageal lacerations at the cardiac orifice of the stomach which had been examined at autopsy. Clinical features emphasize the importance of protracted vomiting as this had occurred in all patients exhibiting lacerations at the cardiac orifice of the stomach; many associated diseases producing the vomiting were present. The lacerations varied from 0.5 cm., to 3 cm., in length and were located at the cardia, extending from the esophagus into the stomach and at times in the cardiac end of the stomach alone, but no cases exhibited lacerations which involved the esophagus alone.

Examination of the histories, in an attempt to clarify the pathogenesis, revealed vomiting as the only common factor. In five cases the history was detailed enough to show that vomiting and retching preceded the appearance of hemorrhage by a sufficiently long time to be of suggestive significance. The original theory of pathogenesis of Mallory and Weiss namely, that the lacerations were produced by violent retching movements forcing gastric contents against a closed cardiac sphincter seemed to be strengthened by these observations.

WILLIAM E. JONES

OBSERVATION OF GASTRIC MOTILITY DURING GASTRIC CRISIS: S. I. Patrick and R. J. Reeves. A.M.A. Arch. Int. Med. 92: No. 6. (Dec.), 1953.

Syphilis of the stomach makes no characteristic clinical picture; it may simulate ulcer or cancer. The most common x-ray findings are hour-glass stomach or *linitis plastica* with or without pylorostenosis. Gummatous disease of the stomach has to be differentiated from gastric crisis due to parasyphilitic disease. Fortunately, tabes or taboparesis rarely occur together with syphilis of the stomach. Therefore, the presence of pupillary, sensory and reflex changes, a positive Wassermann of the spinal fluid and other findings of neurosyphilis exclude organic syphilitic disease of the stomach.

During the tabetic crisis the x-ray changes of the stomach consist of absence of peristalsis and localized and generalized spasms of stomach and bulb. They simulate organic gastric disease just as much as the severe epigastric pain which is usually associated with nausea, vomiting, dysphagia or hiccough.

The x-ray changes completely disappear after a few hours or days and just as suddenly as the pain. Persisting organic changes of the stomach after termination of the gastric crisis indicate ulcer, cancer or other nonsyphilitic disease.

H. B. EISENSTADT

CLINICAL ASPECTS OF JUXTAPILLARY DUODENAL DIVERTICULA: F. Boyer. Deutsche Ztschr. Verdauungs Krank. 14:11-17; 1954.

Three cases of duodenal diverticula close to the papilla of Vater are described. The symptoms included: pain in the upper abdomen with signs of hypoglycemia, such as attacks of sudamina, faintness, paraesthesias of the extremities and bulimia. These are indications of pancreatic involvement. Not only is the pancreatic duct occluded, but a similar fate is frequenly in store for the biliary system in cases of juxtapapillary duodenal diverticula. One case exhibited hepatomegaly with mild jaundice, mild increase of serum bilirubin, as well as a positive Takata-Ara test. These lead to the diagnosis of a low-grade chronic cholangitis with injury to the hepatic parenchyma as a result of mechanical stenosis of the duct.

Another case exhibited a rapid onset of subacute ascending cholangitis. The third case suggested a postoperative biliary condition which is caused by secondary cystic duct obstruction with dyskinesia. An overlooked duodenal diverticulum, however, was the etiological factor. The pathology was located at the ampulla of Vater, resulting in pancreatitis.

Duodenal diverticula located in this locale are not symptomless and generally lead to chronic pancreatitis or involvement of the biliary apparatus. Conservative therapy leads to a poor prognosis because of ulimate development of biliary cirrhosis or of chronic progressive pancreatitis.

REGINALD B. WEILER

THE ANATOMY OF THE PYLORIC CANAL AND THE ETIOLOGY OF INFANTILE PYLORIC STENOSIS: J. Torgerson. Am. J. Roentgenol. pp. 76-80. (Jan.), 1954.

It is believed that this section of the stomach may be conceived of as being constituted by two sphincters; the oral sphincter which corresponds to the sphincter pylori consists of a gastric and duodenal part. The other is a so-called oral sphincter. The pyloric sphincter is continuous on the lesser curvature with a considerable wider oral loop. The pyloric canal often forms the muscular stomachs in lower vertebrae. The significance of the oral sphincter is demonstrated by the fact that the greater swelling of the musculature frequently lies orally to the pyloric valve of the mucosa. In the pyloric valve proper there is frequently no thickening of the musculature at all. The author concludes from this study that pyloric infantile stenosis is due to an abnormal function of the autoplastic closing mechanism of the pyloric canal in which the mucous membrane, through the *torus pyloricus functionalis*, participates to the same extent as the contraction and the stratification of the *propia muscularis*.

J. R. VAN DYNE

THE TWO-COMPONENT MUCOUS BARRIER: Franklin Hollander. A.M.A. Arch. Int. Med. 93:107-120, 1954.

The author discusses his views about why the normal stomach does not digest itself and under what circumstances autodigestion, i.e., peptic ulcer formation in the stomach and duodenum occurs. The stomach is normally protected by a two-component mucus barrier. The first is the nonvital layer of mucus covering the entire gastric inside. The mucus is an effective protection against acid pepsin digestion because of its adhesiveness, cohesiveness, viscosity, adsorptive power and acid neutralizing capacity. The second line of defense is formed by the columnar surface epithelium itself which desquamates when directly attacked by irritative and destructive agents but is able to regenerate itself rapidly thus shielding the delicate glandular structures of the deeper layers. Peptic ulceration occurs when the aggressive acid pepsin factors overwhelm the defense mechanism which may be locally weakened by circulatory, neurotrophic, endocrine, nutritional and metabolic disturbances.

H. B. EISENSTADT

METASTATIC CALCIFICATION AND NEPHROCALCINOSIS FROM MEDICAL TREATMENT OF PEPTIC ULCER: Isidore Snapper, William G. Bradley and Vernon E. Wilson. A.M.A. Arch. Int. Med. 93:807. (June), 1954.

Metastatic calcification occurs in a variety of disorders associated either with an elevation of serum calcium or serum phosphorus or both. The calcium is deposited most

extensively in organs where excretion of acid results in temporary tissue alkalosis, for instance, in the stomach, the lungs and the kidneys. Calcinosis of the subcutaneous and muscle tissues is also common.

The calcinosis observed in ulcer patients, first described by Burnett and associates, is most frequently found after an excessive intake of both alkali and milk, the latter containing large amounts of calcium and phosphorus. The same disorder may be produced by alkalinization alone without milk. A case is reported where the Burnett syndrome appeared after large doses of Alka-Seltzer in a patient whose actual intake of calcium was lower than the recommended daily requirement. A pre-existing renal disease favors the development of calcinosis. This condition can be easily distinguished from hyperparathyroidism because both serum calcium and phosphorus are elevated and an excessive elimination of calcium in the urine is absent. The latter findings explain a frequently negative Sulkowitch's test. In addition, the serum chemistry returns usually to normal after withdrawal of alkali and milk much faster than in hyperparathyroidism. The prevention of the Burnett syndrome requires periodic serum pH and calcium determinations in patients with prolonged conservative ulcer treatment.

H. B. Eisenstadt

VITAMIN E IN THE TREATMENT OF DYSTROPHIC ULCERS OF THE STOMACH: Siegfried Wagner. Aerztl. Forsch. 8:51, (Feb.), 1954.

The dystrophic stomach ulcer, in which serious metabolic disturbances give rise to a lowering of vitality of the stomach wall and thus lead to ulcer, is to be sharply distinguished from the primary peptic ulcer in which the increased stomach activity is the ulcer inducing factor. The different ways in which it can lead to dystrophia are pointed out. The dystrophia primarily affects the muscular system and is manifested in creatonuria. Besides measures to regulate the metabolism of the muscles such as administration of prostigmin and glycocoll there are, above all, the sexual and adrenal cortex hormones which not only halt the creatonuria but also have the capacity to heal the stomach ulcer. The disadvantage of the hormone treatment lies in an early appearance of regressive development following a halting of the pituitary cells. The Vitamin E, in comparison, shows a stimulation of the pituitary diencephalic system, whereby the mentioned metabolic disturbances are removed and vasodilatation, as also capillary germination, is obtained. The trophotrope effect of the tocopherols favors healing of the ulcer by removal of the metabolic damage without setbacks or other disadvantages.

Franz J. Lust

INTESTINES

SURGICAL TREATMENT OF ULCERATIVE COLITIS: Richard B. Cattell, Postgrad. Med. 14:221-225 (Sept.), 1953.

This article is a report of a diagnostic clinic conducted by Dr. Cattell before the 37th Assembly of the Interstate Postgraduate Medical Association. The author has little use for medical treatment and considers psychiatric help of only subsidiary importance. He feels surgery offers the best means of "cure", if wearing a colostomy bag after a mutilating operaton can be so classed.

Dr. Cattell points out that surgical treatment of ulcerative colitis has greatly improved so that the operative mortality has fallen from 20 to 5 per cent. He feels that ileostomy does not shorten life or interfere with sexual potency, provided that no obstruction or *other complication* develops. He describes three operative procedures: Ileostomy, partial or segmental colectomy, and total colectomy. Most patients require colectomy.

The essayist mentions the following indications for surgery: acute fulminating ulcerative colitis, hemorrhage, perforation or fistulae, persistent infectious arthritis, obstruction, malignant degeneration, and intractability. While he denies advising surgery in those cases which can be handled successfully medically, he also states, "If improvement does not occur after 5 to 7 days of medical management, ileostomy should be performed."

Reginald B. Weiler

THE MYENTERIC PLEXUS IN CHRONIC ULCERATIVE COLITIS: K. A. Sorsteen and J. W. Kernohan, Surg. Gynec. & Obst., pp. 335-343 (Sept.), 1953.

The number of ganglion cells in the myenteric plexus at various levels in the colon in 25 cases of chronic ulcerative colitis was compared to those in a like number of normal colons. Approximately a three-fold increase was found in the number of ganglion cells in cases of chronic ulcerative colitis. The number of ganglion cells per cm. in the segmental type was less than in the diffuse variety. The circumferential shrinkage of the segmental type, however, was substantially less and consequently, the estimated total number of ganglion cells was the same. The number of ganglion cells in the uninvolved portions in segmental ypes of chronic ulcerative colitis was usually slightly greater than in the adjacent involved segments. No obvious explanation exists for this increase in the number of ganglion cells in chronic ulcerative colitis. It is possible but improbable the authors think that certain persons are endowed with more than the average amount of myenteric plexus tissue and that chronic ulcerative colitis may develop in some of these persons.

J. R. Van Dyne

CHRONIC ULCERATIVE COLITIS; EFFECT OF A SPECIFIC PSYCHOTHERAPEUTIC MEASURE: Charles G. Craddock, Jr., Psychosomatic Med., 15:513-522 (Sept.-Oct.), 1953.

A case of chronic ulcerative colitis of nine years' duration is discussed. Following a single event, in therapy a dramatic symptomatic improvement followed, lasting several months.

Previous to treatment, in spite of many overwhelming traumata including loss of loved one, this patient had been unable to discharge any of her affects verbally and could only obtain partial relief through "organ language" expression such as the colitis. It is interesting that the improvement occurred before any interpretations. The remission seems to have been due to the ther-

apeutic setting which allowed her to verbally and emotionally discharge the pent-up affects. She was then able to work through the grief; which in turn opened up a channel for associated repressions to flow into consciousness. The reconstruction which followed is regarded as "replacement therapy".

REGINALD B. WEILER

LIVER AND BILIARY TRACT

II. PSYSIOLOGIC STUDIES IN CASES OF STRICTURE OF THE COMMON BILE DUCT: TWENTY-EIGHT YEARS SURVEY WITH DATA ON 254 PATIENTS: W. Walters, Ann. Surg., 138:609 (Oct.), 1953.

Two hundred fifty-four cases with postoperative stricture of the common bile duct seen between 1924 and 1951 are reviewed. The necessity for an unrelenting search for a usable proximal portion of a strictured or severed duct is stressed. Anastomosis was carried out between portions of the bile ducts themselves, the common bile duct and the duodenum, and the hepatic ducts and the duodenum, choledochoduodenostomy being the most frequent. The author feels that all patients are entitled to reexploration regardless of the number of previous operations, unless liver damage is so great and general condition so poor that survival of surgical trauma is not likely. It is not felt that reflux of intestinal contents into the bile ductal system causes any infection but that rather any cholangitis that occurred was due to stricture at the site of anastomosis. To prevent such a stricture the use of a prosthetic appliance to splint the anastomosis for a period of six to nine months following surgery is advocated.

WILLIAM E. JONES

BOOK REVIEWS FOR GASTROENTEROLOGISTS

ATLAS OF EXFOLIATIVE CYTOLOGY: George N. Papanicolaou, M.D., Ph.D. Loose leaf, 36 colored plates. Published for Commonwealth Fund by Harvard University Press, Cambridge, Mass., 1954. Price $18.00.

In this loose leaf volume by Dr. Papanicolaou, the illustrations depicting the abnormal cells are beautifully reproduced. Studying the various sections, the reviewer is amazed with the amount of valuable and instructive material the author included in the atlas. For instance, 12 plates relate to the female genital system; 4 to the urinary organs; 5 to the respiratory system and 5 to the gastrointestinal tract; 2 to the pleural, peritoneal and pericardial exudates; 2 to the breast, etc.

The author agrees that the cytological method of diagnosis is still a screening process and does not compare with actual tissue biopsy.

However, tissue biopsy and cell cytology may well go hand in hand as a diagnostic aid in the early recognition of early cancerous changes.

1952 YEAR BOOK OF RADIOLOGY: Edited by Fred J. Hodges, M.D., John F. Holt, M.D., Harold W. Jacox, M.D. and Vincent P. Collins, M.D. 416 pages, illustrated, with a cross and authors index. The Year Book Publishers, Inc., Chicago, Ill., 1952. Price $7.50.

This volume is divided into two sections: Radiologic Diagnosis and Radiation Therapy.

As all other year books, this volume consists of pertinent abstracts of the world literature dealing with the latest developments in new apparatus technic and therapy.

No physician, no matter how prolific a reader may be, has the time and opportunity to scan the vast amount of material appearing in medical journals dealing with the various aspects of roentgenology. Here in this book, the roentgenologist, the general practitioner and specialist will find a comprehensive resume of what he is interested in.

It is highly recommended.

MANUAL OF PROCTOLOGY: Emil Granet, M.D., Lecturer, Graduate School, Columbia University, Visiting Surgeon (Proctology), Seaview Hospital and Associate Surgeon (Proctology), French Hospital, New York, N. Y. 346 pages, profusely illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1954. Price $7.50.

The book is divided into 18 chapters, a cross index and references. It is written for the general practitioner rather than the specialist in rectal surgery.

The chapters on general therapy and pediatric proctology should interest the general practitioner, as well as the pediatrician. It is clearly and concisely written and the illustrations are self-explanatory.

The chapter on hemorrhoids, surgical and nonsurgical, ably describes what the nonspecialist can do to alleviate hemorrhoids before he refers the patient for surgery.

Tumors, benign and malignant, the various colitides, pruritus ani, medical and surgical treatment, proctalgias, anal rectal dyscrasias, and a host of miscellaneous conditions affecting the bowel and anal canal, etc., are concisely discussed and treatment outlined.

It is highly recommended as a valuable reference book for the busy physician.

DEVELOPMENTAL DISORDERS OF MENTATION AND CEREBRAL PALSIES: Clemens E. Benda, M.D., Director of Research and Clinical Psychiatry, Walter E. Fernald State School, Waverley, Mass., Instructor in Neuropathology, Harvard Medical School, Assistant in Psychiatry, Mass. General Hospital, Lecturer, Postgraduate Seminar, Mass. Department of Mental Health. 565 pages, 102 illustrations. Grune and Stratton, Inc., New York, N. Y., 1952. Price $12.75.

The author of this volume is an experienced neuropsychiatrist and teacher and has published several books.

He states "the purpose of science is to serve the interests of humanity. If this book enables others to take up where the author

has left off, its objective has been accomplished."

The author discusses the inadequacy of present day concepts of mental deficiency and mental illness, genetic and exogenous factors, classification of antenatal disorders, developmental disorders of the spinal cord, developmental disorders of the cerebellum, cranial dysostoses, the hydrocephalies and microcephalies are satisfactorily presented. Ectodermal dysplasias and neoplastic malformations, cerebral palsy, birth injuries and the lipoidoses also gargoylism, Morquio's disease, Gregg's (1924) disease (hypertelorism), degenerative diseases and childhood schizophrenia ("Morbus Bleuler"), are instructively and informatively discussed.

Hurler-Hunter's syndrome (chapter 15) is a metabolic disorder which affects: (1) the liver, lungs, heart, spleen and blood; (2) the mesodermal elements in the nervous system, meninges and vascular system; (3) brain and spinal cord and (4) the membraneous tissues of the eyes. Hunter (1917) and Hurler (1919), also Thompson (1900) and Berkhan (1907) described cases of gargoylism ("Hunter-Hurler syndrome", pages 407-445). Glycogen storage disease (Von Gierke, 1929), "Foelling's disease" (1934) or phenylpyruvic oligophrenia and Laurence-Moon-Biedl syndrome, "Gaucher's disease" (lipoid cell spleno-hepatomegaly), etc., are briefly but adequately discussed.

This work will be of interest to pediatricians, clinicians, neuropsychiatrists and students of the subject.

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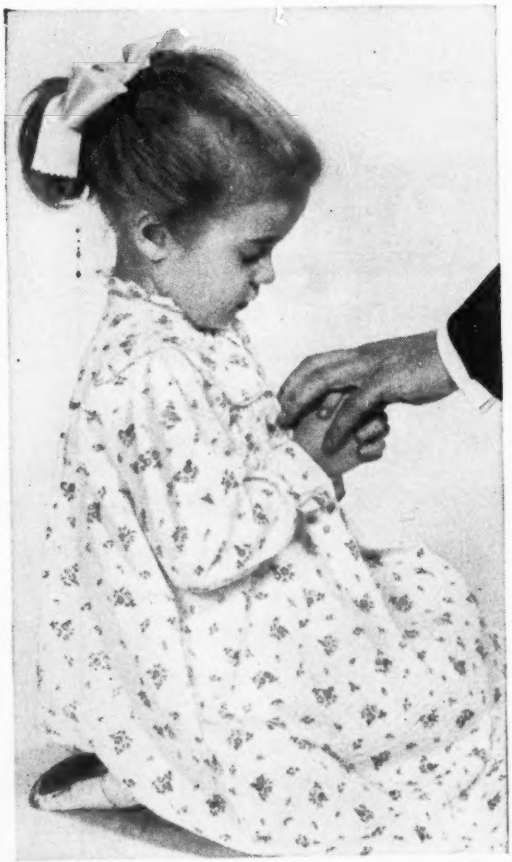

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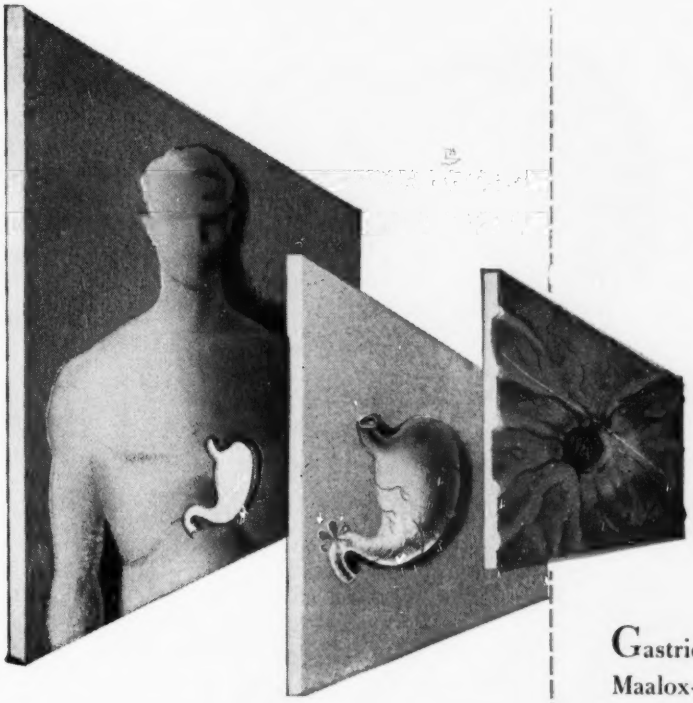

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(1) Sweatman, C. A.: J. So. Carolina M. A., 49:38, 1953. (2) Marks, M. M.: Am. J. Dig. Dis. 18:219, 1951. (3) Hamilton, H., in Trans. 5th Am. Cong. Obst. & Gyn., Mosby, 1952, p. 69. (4) Burnikel, R. H., & Sprecher, H. C.: Am. J. Dig. Dis. 19:191, 1952. (5) Marks, M. M., Personal Communications, 1952-53.

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1. ROGERS, M.P., AND GRAY, G.L.: AM. J. DIGEST. DIS. 19:180 (JUNE) 1952.

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*Sherber, D. A.: The Control of Bleeding, Am. J. Surg. *86*:331 (Sept.) 1953.

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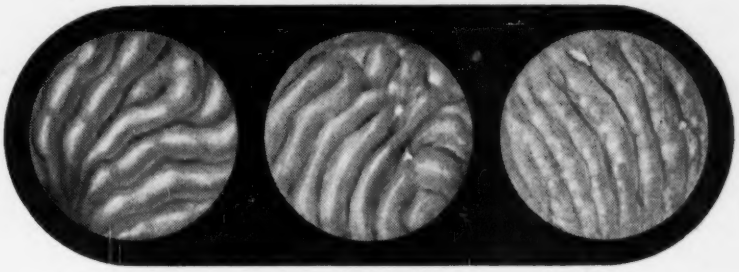

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References: 1. Hollander, F.: Arch. Int. Med. *93*:107 (Jan.) 1954
2. Deutsch, E.: Scientific Exhibit, Gastroscopy, Interim Session A.M.A., St. Louis, December, 1953

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